Copy 2. Periodicals 67

UNIVERSITY OF TORONTO MEDICAL JOURNAL

GASTROENTEROLOGY

Editorial.

Report of the President of the Medical Society. J. Morphet, 5T7 2

Biliary Tract Disease. R. I. Hector, 5T7 4

Functional Disorders of the Gastrointestinal Tract. J. C. Deadman, 5T7 11

Chronic Diarrhoea: A Problem in Diagnosis. M. A. Tyber, 5T8 15

Peptic Ulcer. N. L. Kramolc, 5T7 17

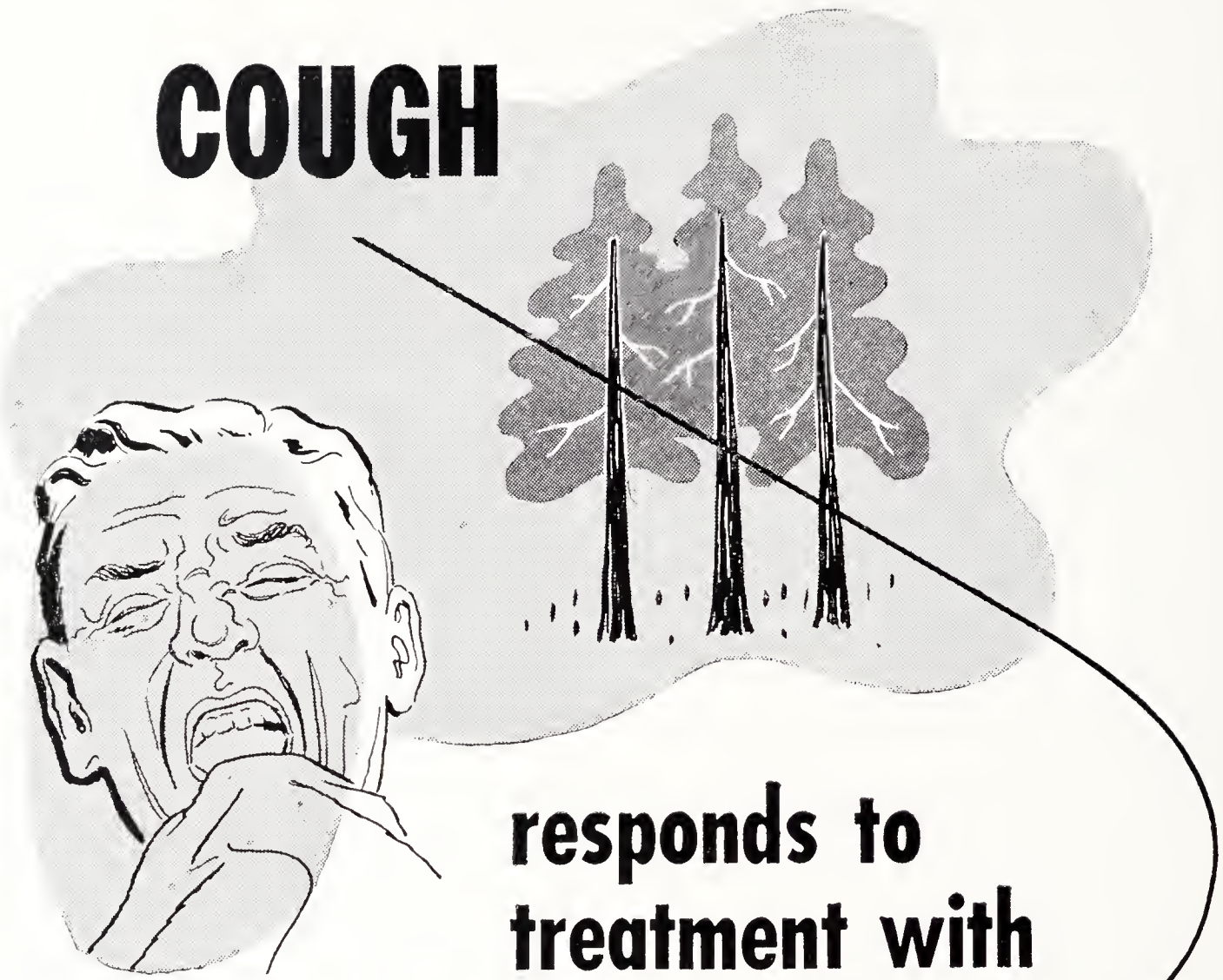
Abstracts 22

Addresses of the Graduating Class of 1956 25

NOVEMBER, 1956

VOLUME XXXIV NUMBER 1

COUGH



responds to treatment with "PINOCODEINE"(N)

Each fluid ounce contains:

Pinus strobus	32 gr. (2.1 G.)
Prunus virginiana	32 gr. (2.1 G.)
Sanguinaria canadensis	4 gr. (0.25 G.)
Populus balsamifera	2 gr. (0.12 G.)
Chloroformum	1 min. (0.6 cc.)
Codeinae phosphas	1 gr. (65 mg.)

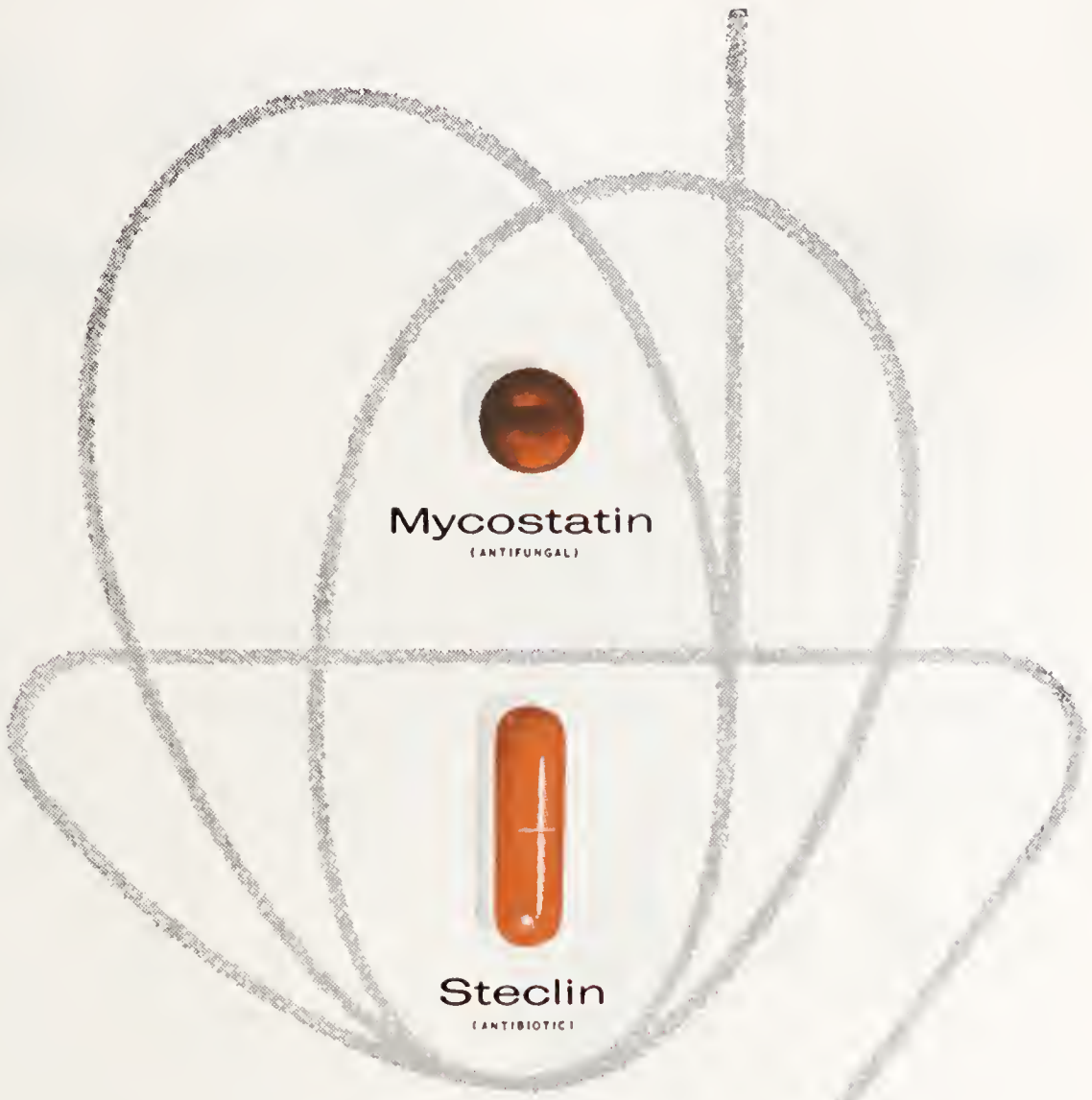
(N) *Oral narcotic prescription permitted.*

DOSAGE: One or two drams every 4 hours.

Packaged in 16 fluid ounce bottles.

Frosst

Charles E. Frosst & Co.
MONTREAL CANADA



The only tetracycline with added antifungal prophylaxis

Mysteclin

(SQUIBB TETRACYCLINE-NYSTATIN)

"...any patient requiring broad spectrum therapy surely deserves the added protection of Mysteclin against intestinal moniliasis."

SQUIBB

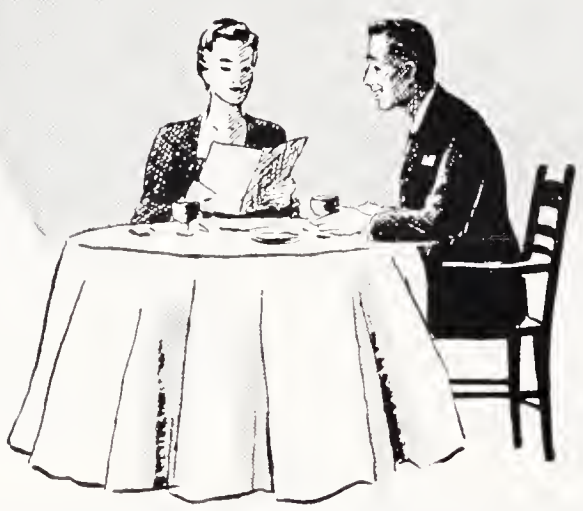
Each capsule contains 250 mg. tetracycline + 250 M.U. nystatin.

Supply — Bottles of 12 and 100 capsules.

Write: E. R. Squibb & Sons of Canada Ltd., P.O. Box 599, Montreal, P.Q.

MYCOSTATIN, STECLIN AND MYSTECLIN ARE TRADE-MARKS OF E. R. SQUIBB & SONS OF CANADA LTD.

to depress appetite - safely



- protect against nutritional deficiencies
- make dieting easier

"NEGADINE" BRAND

Capsule No. 681 "Frosst"

Each capsule contains:

d-amphetamine sulphate	5 mg.
Ferrous sulphate (exsic.)	50 mg.
Copper sulphate	2.5 mg.
Manganese carbonate	0.3 mg.
Vitamin A palmitate	1000 I.U.
Vitamin D	1000 I.U.
Calcium phosphate (dibasic)	140 mg.
Thiamine hydrochloride	1 mg.
Riboflavin	1 mg.
Ascorbic acid	25 mg.
Sodium iodide	0.2 mg.

Packaged in bottles of 25, 50 and 100 capsules.

DOSAGE

One capsule half an hour before breakfast and one capsule half an hour before lunch.

Frosst

Charles E. Frosst & Co.
MONTREAL CANADA

HOW VAGISEC LIQUID

PENETRATES

RECESSES OF VAGINA
AND EXPLODES
TRICHOMONADS
OFTEN MISSED

TOO OFTEN AN ORDINARY trichomonacide fails to cure vaginal trichomoniasis because it has little or no effect on parasites that are not on the surface.[1] Trichomonads burrowed deeply into the roughened mucosa survive and set up new foci of infection. In fact, even a few hidden trichomonads remaining after treatment can cause acute exacerbations. With VAGISEC* liquid and jelly you can overcome this most troublesome problem.

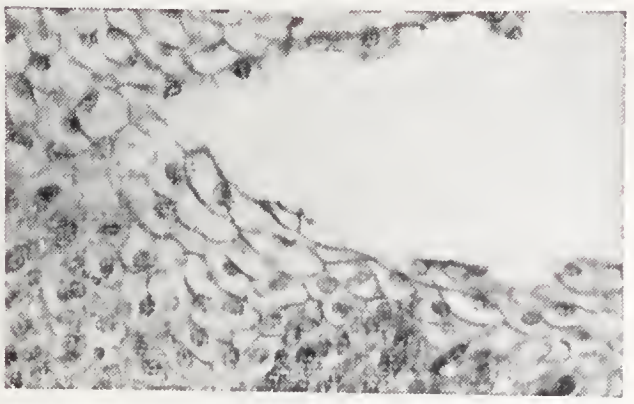
Penetrates thoroughly — This unique trichomonacide rapidly dissolves mucinous materials, fats and blood clots.[1] It penetrates cellular debris and reaches trichomonads deep in their hiding places.

Explodes trichomonads — VAGISEC liquid actually explodes trichomonads within 15 seconds after douche contact.[2] Two surface-acting agents and one chelating agent combine to weaken the cell membrane, to remove the waxes and lipids, and to denature the protein. Then the parasite imbibes water, swells and explodes.

Proves highly effective—With the Davis technique† you can now rid patients of "trich," even cases that have resisted other treatment. VAGISEC liquid was developed as "Carlendacide," by Carl Henry Davis, noted gynecologist, and C. G. Grand.[1] Clinical trials by more than 150 physicians show better than 90 per cent success.[3]

Use liquid and jelly — In the Davis technique, VAGISEC liquid is used in office therapy, and liquid and jelly are prescribed for home use.

Photomicrograph of section of epithelium of normal vaginal mucosa, enlarged 750 times, shows uneven surface where trichomonads hide. VAGISEC *penetrates surface and explodes organisms in hard-to-reach areas.*



Office treatment — Wipe vaginal walls dry with cotton balls, then wash thoroughly with a 1:100 dilution of VAGISEC liquid. Remove excess fluid with cotton balls. Dr. Davis recommends six treatments.

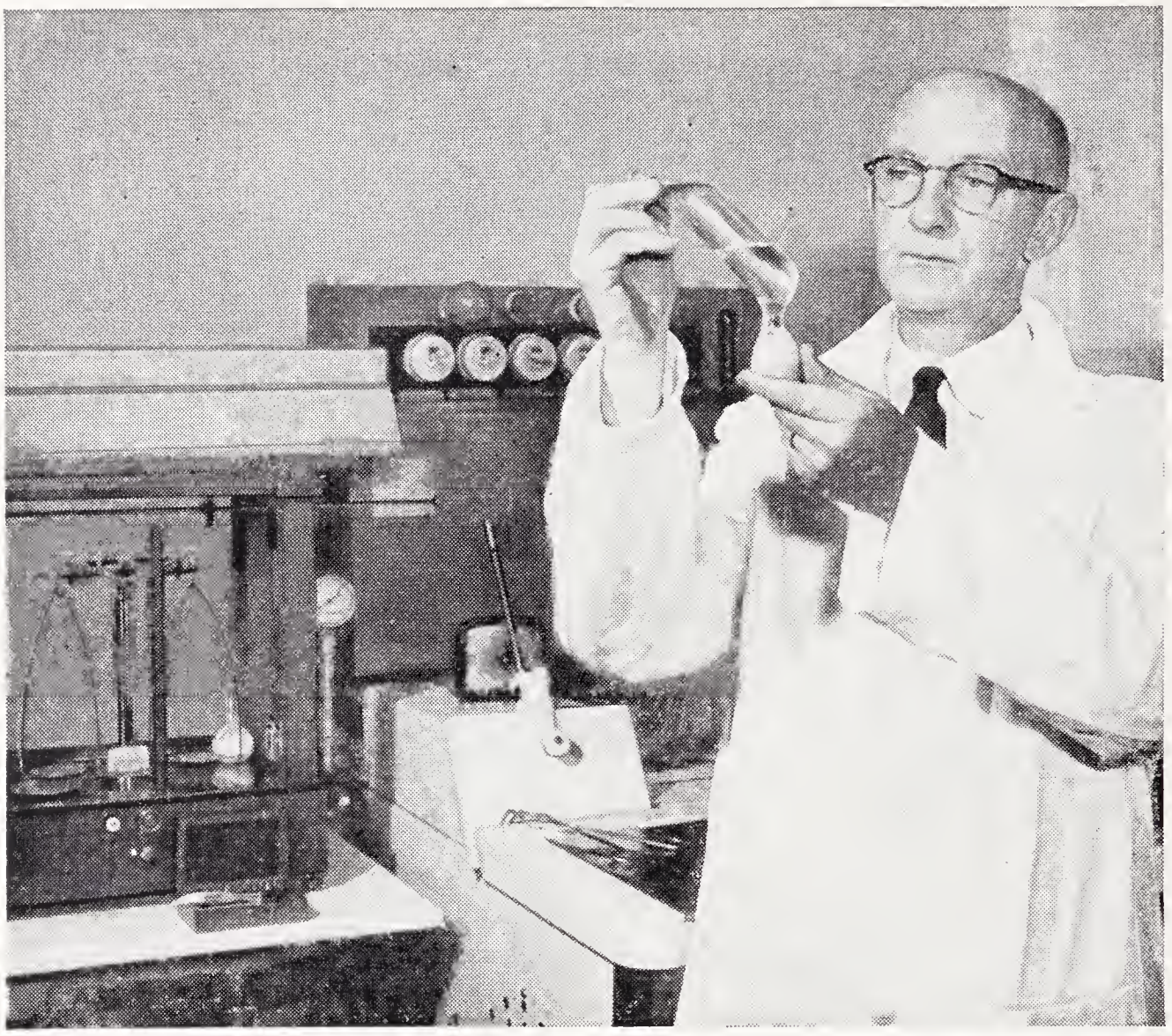
Home treatment — Patient douches with VAGISEC liquid every night or morning and then inserts VAGISEC jelly. Home treatment is continued through two menstrual periods, but omitted on office treatment days. Douching is contraindicated in pregnancy.

Active ingredients in VAGISEC liquid: Polyoxyethylene nonyl phenol, Sodium ethylene diamine tetra-acetate, Sodium dioctyl sulfosuccinate. In addition, VAGISEC jelly contains Boric acid, Alcohol 5% by weight.

References: 1. Davis, C. H., and Grand, C. G.: Am. J. Obst. & Gynec. *68*:559 (Aug.) 1954. 2. Davis, C. H.: J.A.M.A. *157*:126 (Jan. 8) 1955. 3. Davis, C. H.: West. J. Surg. *63*:53 (Feb.) 1955.

*Trade-mark
†Pat. App. for

JULIUS SCHMID (Canada) Ltd.
32 Bermondsey Road
Toronto 16, Canada



W. D. LAVERTY—25 years milk laboratory experience—conducts quantitative butterfat test on evaporated milk using the Majonnier method and testing equipment.

Just one of a number of checks

During processing, Farmer's Wife Formula Milks constantly undergo a number of tests in the plant and laboratory. Even after vacuum sealing in the most modern type of container, the quality of the individual can and contents is verified. It is this kind of constant check that leads doctors, paediatricians and hospitals from coast to coast to confidently recommend Farmer's Wife Formula Milks.

- Evaporated whole milk
- Concentrated skimmed milk
- Concentrated partly skimmed milk

Farmer's Wife

COW AND GATE (CANADA) LIMITED

7-6

"Specialists in the Preparation of Milk Foods for Infant Feeding"

UNIVERSITY OF TORONTO
MEDICAL JOURNAL

ESTABLISHED DECEMBER 1923
AN UNDERGRADUATE PUBLICATION

VOLUME XXXIV NUMBER 1
TORONTO, NOVEMBER 1956

Editor-in-Chief..... J. C. Deadman, 5T7

Associate Editor..... A. C. Strickler, 5T8

Assistant Editors..... J. A. Lowden, 5T7
M. A. Tyber, 5T8

Advertising Manager..... P. C. F. Moore, 5T9

Circulation Manager..... P. B. Dent, 6T0

Staff Advisor..... C. B. Brown, MD.

This Journal is published six times a year from November to April, by the Medical Society of the University of Toronto, to serve primarily as a medium for student expression.

Subscription Rates: $3.50 for one volume of six consecutive issues. Single copies 65¢.

Information to Contributors

Manuscripts including references and footnotes should be typewritten double spaced on one side of the paper. References should be kept to a minimum and should be cited numerically in the order in which they appear in the text.
Illustrations are included by arrangement with the Editor. Drawings should be done on white paper in black ink.

Final copy date is the 15th of the month before the issue concerned.

Material printed in this journal is covered by copyright. Articles are accepted for consideration with the understanding that they are contributed for publication in this journal only.

Correspondence should be addressed to Room 114, Medical Bldg., University of Toronto, Toronto 5, Ontario.

Authorized as Second Class Mail, Post Office Department, Ottawa.

EDITORIALS

Policy This Year

The Journal is being launched on another year of publication with, we hope, fresh enthusiasm and interest. Each year we have similar problems and make some of the same mistakes. It is our aim this year to present a journal with articles of practical value and interest to the students, and also of interest to our other subscribers. In any journal whose policy it is to present review articles exclusively, the tendency sometimes seems to be either to treat subjects already well covered by textbooks which every student may be expected to have, or, to treat subjects which are rather remote from both the experience and the interest of the reader. Articles for such a journal should be stimulating reading, and carry one beyond the facts normally covered and required, either for exams or in practice, without bogging down in a welter of detail about subjects seen once or twice in a lifetime. It must be appreciated that for many students, this is the only outside medical reading which they do.

Our policy this year will be to treat some of the more interesting aspects of the main organ systems, following, as before, the idea of assigning to each issue a topic. We shall also include case reports and abstracts in every issue. While we hope these articles will be useful for studying, it is our aim that they should be much more than this, and stimulate a little thought on related subjects over and above that required in day-to-day work or for student examinations.

Many students, we feel, do not take sufficient interest in many of the extra-curricular activities which make the difference between being trained and being educated. This is a perennial problem and one which has been dealt with repeatedly. It is mentioned only to point out that we are attempting to get nearly all student articles this year, a policy not always followed in the past. This, we hope, will not only give those who want to write the opportunity of doing so, but spur some others who have never really considered breaking into the ranks of the "greats", the chance to do so. This we hope at any rate.

Difficulties

We do not wish to seem discouraged by the student, not in the first premedical year who asked what the Journal was, and when told that it was published by the students of Medicine, was very surprised and said "Gee I guess I should read it, I never bothered picking them up last year," but it does seem that there has been a breakdown in Public Relations somewhere.

J.C.D.

Report of the President of the Medical Society

Two questions asked this morning in *Martin's Manor* prompted the substance of this article. The first was "what has the Medical Assembly been doing this year?" and the second, "Don't you think that Jack Martin has got more fellows through this course than all the Professors put together?"

In answer to the first, I am happy to report that the Society as a *whole* has been busy, not just the Assembly. Perhaps the statement of Bill Hunter (President 5T6) that "Medical spirit need only be encouraged, not whipped" is in the process of being realized. At this time, half way through the present assembly's term of office may I applaud: Mike Allen's May tribute to Doctor Grant; Murray Zane's 'show' float; Lalene Putz's unique Freshman Orientation; Charlie Babin's tremendous Freshman Reception (with added appreciation to Doctors MacFarlane, Farquharson, Wightman, Hamilton and Anderson) Andy Baine's energetic Arts and Letters program; Joe Stipec's Duncan Room persistence; Dave Price's sound Assembly judgment; Lionel Chisholm's financial finesse; Sylvia Fine's Secretarial thoroughness; Louise Chevalier's smoothly-running M.W.U.A.; Jimmy Hamilton's potential Reed Trophyers and of course John Deadman and the excellent Journal which you hold in your hand. Holly King 5T7 and Larry Wilson 6T2 make invaluable Presidential 'Ends' on any team. Also we are extremely fortunate in having on our Assembly this year's S.A.C. president, Gord Forstner. George Langmyhr and Gord Perkin are making C.A.M.S.I. much more intelligible to us and the Wait-Tator combination are approaching E.A.C. problems with wisdom. On the horizon we look forward to Daffydil and the Staff-Student Smoker, Bob Roe and Jim Gordon guiding them. The Meds At-Home is to be held on Valentine's Day in February. We hope it will prove even more successful than the Meds/POTS Fall Dance which Ted Morgan also organized so well. And we owe much in the way of sound leadership to Dr. Ross Doyle (5T3) who is our chairman this year. This is a bare outline of what we have been doing.

The second of our two questions is answered in the affirmative. Perhaps four hundred years from now when there is no Jack Martin in this Medical School, something of the "Art of Medicine" may be absent in the graduating student. Bacteriology may be taught in the Banting, Histology in the Anatomy Building, but our course in Interpersonal Relations is taught in only one place, and the Clinician in this subject does not teach, he personifies. Although Mr. Martin is only recently an Honourary Member of the Medical Society, he has adopted us years ago. He embodies the old saying that it is really the little things in life that count; the coffee and joke before an oral, the refreshed incentive in being called (somewhat prematurely) "Doctor", the advice and reassurance given on innumerable personal problems. Caffeine alone does not convey such genuine interest in our well-being. He doesn't preach, but somehow we come to learn what tolerance, humility and understanding really mean. Perhaps these are the keys to the "Art of Medicine".

In essence Jack Martin is the guardian of the Medical Society, a much appreciated and indispensable friend of the student. "Hardly seems enough to ask, Doctor. . . ."

JOHN MORPHET

BILIARY TRACT DISEASE

R. I. HECTOR

ANATOMY AND EMBRYOLOGY

THE gall bladder is a thin walled, musculomembranous organ about 10 cm. in length which lies attached by loose connective tissue to the inferior surface of the right lobe of the liver, and it is entirely covered with peritoneum except for this aspect. The fundus usually reaches the anterior border of the liver and when distended may be felt against the anterior abdominal wall. Important regional structures are the stomach, duodenum, pancreas and transverse colon.

The cystic duct runs postero-medially to join the common hepatic duct formed in the depth of the transverse fissure by the ducts from the right and left lobes. The union of these forms the common bile duct which passes inferiorly in the edge of the lesser omentum, behind the first part of the duodenum to enter the second part at the ampulla of Vater about 2½ cm. below the pylorus.

The cystic artery, which usually arises from the right hepatic branch of the common hepatic just after its bifurcation, runs close to the cystic duct and supplies both the duct and the gall bladder. The course and relations of this artery are subject to wide variations, hence all these structures must be clearly visualized at operation.

Most of the lymphatic drainage is to the cystic gland which lies close to the junction of the cystic and common ducts; some lymphatics directly enter the liver. Recently, lymphatic connections between the gall bladder and pancreas have been demonstrated.

During the fourth week of intrauterine life, a solid bud of tissue arises from the ventral aspect of the primitive foregut to form the liver and duct system. Any arrest or abnormality of development produces congenital anomalies such as atresia of the bile ducts, choledochal cyst, double gall bladder or aberrant ducts.

PHYSIOLOGY

Almost the entire daily flow of bile from the liver (500–1,000 cc) is carried to the gall bladder which concentrates and stores the bile, through the absorption of water and inorganic salts. From here it is supplied on demand during digestion. The contraction of the gall bladder and the expulsion of bile appears to be under the control of a hormone, cholecystokinin, which is released from the mucosa of the duodenum and small intestine upon stimulation by food, in particular, fat.

The main components of bile are the pigments, the alkali salts of bile acids, inorganic salts, cholesterol and mucoprotein. The most important role of bile is in the digestion and absorption of fats and fat-soluble vitamins.

SYMPTOMATOLOGY

Pain

Almost always associated with gall stones, biliary colic is the most important and characteristic feature of biliary tract disease and it is produced by spasm of the smooth muscle of the biliary tract along with an abrupt rise of intramural pressure. It is usually a very severe disabling pain which builds up rapidly, is maximal for a period of 10–40 min., subsides gradually over a period of hours and is frequently followed by a prolonged period of aching in the epigastrium. It is a steady pain without much fluctuation, and appearing first in the epigastrium most commonly on the right side, it may pass along the right costal margin to the mid-back or the inferior angle of the right scapula. Com-

mon variants of this distribution are that the pain may pass chiefly to the left upper or right lower abdominal quadrant. The patient during the attack is restless, and the attacks have a tendency to occur within 3–5 hours after meals. Pain of a similar distribution, but much less severe in nature, may be caused by inflammation alone.

Jaundice

This sign is only seen in 25% of cases and when present, it is regurgitation in type and fluctuating in intensity. The rate of appearance and its intensity vary greatly from case to case.

Fever and chills

These appear to be caused by a combination of biliary stasis and infection, the chills probably representing bouts of transient bacteraemia. Temperature, however, may be very misleading for 75% of patients never have an oral temperature exceeding 99.0° F.

Disorders of the GI tract

Nausea, vomiting, flatulence and constipation associated with biliary pain are of clinical importance, but when occurring without it are much more commonly due to other unassociated disturbances. Reduced or absent bile flow following obstruction, rapidly impairs digestion and absorption of fats, so that 10–40% of the fat intake may be lost in the stool and a much reduced absorption of vitamins A, D, E, and K occurs.

Bleeding

Unless associated with hypoprothrombinaemia, bleeding is rarely of clinical importance in biliary tract disease.

DIAGNOSTIC PROCEDURES

(a) Simple X-ray; a negative flat plate of the abdomen is of no significance for 50% of gall stones are radiolucent.

(b) Cholecystography; This test, dependent upon the ability of the gall bladder to concentrate radio-opaque dye excreted by the liver, is most useful in the diagnosis of cholelithiasis.

(c) Duodenal drainage; the presence of cholesterol crystals in the aspirated material is pathognomonic of biliary tract disease, but the test is difficult to perform and seldom used.

(d) Operative cholangiography; Radio-opaque dye injected directly into the bile ducts is invaluable in the localization of obstruction or in the demonstration of patency of the ducts.

DIFFERENTIAL DIAGNOSIS

Biliary tract disease must most commonly be differentiated from other causes of the acute abdomen. Acute appendicitis, perforated peptic ulcer, renal colic, intestinal obstruction, mesenteric thrombosis and tabetic crises must be considered and on occasion a preoperative diagnosis will be impossible. Right basal pleuro-pneumonia or diaphragmatic pleurisy may be suggested in taking the history, but physical examination will usually reveal the diagnosis. Coronary thrombosis may also manifest itself with biliary tract-like symptoms and it must be remembered here that similar electrocardiographic changes may result from acute cholecystitis.

Jaundice, when present, requires that it be differentiated from carcinoma of the head of the pancreas, chronic pancreatitis, intrahepatic jaundice and hemolytic crises.

It should also be noted that any elderly person with an unexplained fever and leucocytosis should be suspected of having biliary tract disease and a careful search made, for signs and symptoms may be minimal.

PATHOGENESIS

Cholecystitis

Existing evidence seems to indicate that the common cause of cholecystitis is a chemical irritation from overcon-

centration of bile salts or other bile constituents following partial or complete obstruction of the cystic duct. Associated spasm and edema of the ducts may also involve the cystic artery and ischemic damage be added to the picture. Occasionally, bacteria are isolated from the inflamed wall, but evidence suggests that these are secondary invaders.

Cholelithiasis

In experiments in coronary artery disease, it has been shown that the incidence of gall stones in a large group on a low fat diet was under one percent whereas the incidence in the control group was eight percent. It is suggested that a high fat diet produces an increase in cholesterol and a decrease in the bile salts in the bile with resultant precipitation of cholesterol and calcium bilirubinate to form gall stones. The low incidence of cholelithiasis in some Asiatic countries may be related to their low fat diets.

GALL STONES

In a large series of autopsies, the general incidence was 9.9% with the incidence in white females over the age of twenty being 21% and that in white males over the age of twenty 7.7%. They are very uncommon in the negro race. The individual may have one stone or he may have hundreds. Single stones are large and round or elliptical; multiple stones tend to be small and faceted. Pure pigment or pure cholesterol stones are rare, and about 50% contain enough calcium to make them radio-opaque.

Signs and symptoms

Half the people with gall stones have no biliary symptoms but the remainder suffer from attacks ranging from mild epigastric distress to a full blown colic. The attacks of colic occur intermittently in response to calculi in the pouch of Hartman or passing down the cystic and common ducts, and they are not necessarily associated with acute cholecystitis. The attacks may come on at any time of the day or night, but most commonly occur from 9 p.m. to 3 a.m., and between them the patient feels perfectly well.

Course and indication for surgical treatment

The possible complications being acute and chronic cholecystitis, choledocholithiasis, cholangitis, hepatic abscesses, biliary cirrhosis and biliary fistulae, it is evident that patients with one or more attacks of biliary colic should be treated by cholecystectomy for there is no medical means of cure, and one should attempt to control as early as possible a condition which with advancing age becomes more and more precarious.

Contributing Factors

Important factors appear to include pregnancy, for many women relate their first symptoms to a pregnancy. Menstrual function may also be associated with onset of symptoms and some women have regular recurrences of symptoms associated with the menses. Persons on a prolonged diet with a marked weight loss will occasionally develop symptoms and this is thought to be due to the increase in lipid metabolism in persons burning their own body fats. Infection and stasis of bile have also been incriminated, but no conclusive evidence has been found as yet.

Treatment

When associated only with fatty indigestion, surgery should never be resorted to for cure since it is seldom successful. Medical management with diet, antispasmodics and other symptomatic therapy is indicated here. The patient with silent stones should be carried without specific therapy for there is adequate evidence that surgical therapy carries with it a risk as great as the natural course of the disease. There may be rare

exceptions for example, the patient with a functional gall bladder and multiple small calculi.

ACUTE CHOLECYSTITIS

Associated with cholelithiasis in the great majority of cases, this condition appears to be caused by the sudden impaction of a calculus at the mouth of the cystic duct with a resultant combination of chemical irritation and ischemic necrosis. Rarely, a primary bacterial infection may be the cause.

Symptoms

Pain, moderate or severe, and fever spiking to 101°F at least once in every 24 hours are the outstanding symptoms. Jaundice is rarely present and when so, is usually mild and transient, associated with oedema and spasm of the ducts draining the liver.

Signs

Abnormalities in the right upper quadrant, ranging from slight muscle guarding to marked rigidity with a tender, indefinite mass palpable in about half the cases, are the main signs on physical examination.

Course

Many attacks subside spontaneously in two to four days, but in 40% of cases, it may progress with danger of empyema, gangrene, or perforation. Uncommonly, the stone may erode the wall of the gall bladder producing a biliary fistula.

Treatment

Early surgical intervention is recommended by most schools; certainly if there is a progression of signs and symptoms some operative intervention is necessary, perhaps just cholecystotomy. One must remember that there is an added mortality with such an emergency procedure, as edema may obliterate landmarks and make the surgeon less precise. However, after the proper preparation of the patient, cholecystectomy appears to be the procedure of choice, although cholecystostomy may prove lifesaving in the poor risk patient.

CHRONIC CHOLECYSTITIS

Previous attacks of acute cholecystitis associated with cystic duct obstruction or continued partial obstruction account for many cases of chronic cholecystitis, although rarely infection or reflux of pancreatic enzymes may be the etiological agent.

Clinical Picture

Occurring most commonly in the middle aged female, the most common symptom is a continued epigastric distress varying from a flatulent dyspepsia associated with dull right upper quadrant pain to biliary colic. Examination usually reveals tenderness to fist percussion over the involved region and a positive Murphy's sign. Fever is uncommon.

Treatment

If the diagnosis can be confirmed, cholecystectomy is the treatment of choice. If the psychogenic element appears to be preponderant, such a procedure is not justified for the symptoms are most likely to recur after operation.

TORSION OF THE GALL BLADDER

This is a very uncommon condition, associated with unusual anatomical relationships between the gall bladder and the liver. It presents with the same picture as that of acute cholecystitis and its only importance is that the patient may die while one awaits the normal resolution of the latter disease.

CHOLEDOCHOLITHIASIS

Common duct stones occasionally originating within the bile ducts, enter from the gall bladder, and they may remain in the ducts or pass through

them, in either case producing a wide range of symptoms.

Clinical picture

If the stones become impacted, they produce biliary colic, intermittent fever and a fluctuating jaundice. All of these vary with the completeness of the obstruction and the variations in the clinical picture may be extreme.

Treatment

The treatment is essentially surgical, although symptomatic therapy with antispasmodics may be of value in the relief of pain during the acute stages. When the patient's condition permits the common duct must be explored for stones and a cholecystectomy is usually performed at the same time. Prior to the removal of the T-tube, over which the common duct is closed, a cholangiogram should be performed to ensure patency of the bile ducts.

BILIARY FISTULAE

An uncommon sequel to acute cholecystitis and biliary surgery, these are seen more commonly in females from 50–70 years of age. Cholecystoduodenal, cholecystocolic and choledochoduodenal are the common varieties, but they may communicate with almost any abdominal or thoracic structure.

Etiology

90% result from complications of gall stones, 6% with perforating ulcers and 4% with carcinoma of the biliary tree.

Clinical picture

In internal fistulae, the symptoms are those of biliary tract disease, but intestinal obstruction due to the stone, or heamatemesis and melena may also occur. In external fistulae, openings occur near the umbilicus or on the right upper quadrant of the abdomen, and all abscesses and sinuses in this region should be carefully examined for this lesion.

Diagnosis

Air insufflation of an external fistula, or a routine gastric series will frequently show the biliary tree and the fistulous tract.

Treatment

Cholecystectomy and excision of the tract with closure of the opening in the intestine is indicated.

TUMORS OF THE GALL BLADDER

Benign tumors are uncommon and usually asymptomatic. They may produce symptoms resembling chronic cholecystitis.

Carcinoma, by far the most common malignant tumor of the gall bladder, is seen in 1.5% of cholecystectomies and occurs mainly in females from 50–70 years. The preoperative diagnosis of this condition is seldom made and the symptoms are those of a cholecystitis.

Etiology

The presence of calculi appears to be very important for in almost every case, there is an associated cholelithiasis.

Pathology

Two main groups are recognized; adenocarcinoma and epidermoid carcinoma—rarely a squamous cell carcinoma is seen. The wall of the gall bladder is thick, hard and contracted and frequently mistaken for chronic cholecystitis. The tumor metastasizes early with direct spread to the liver and extension along the bile passages.

Treatment

Surgery is the only hope, but the 5 year survival is very low, being about 2%.

DISEASE OF THE BILE DUCTS

Choledochal cyst

A very rare condition of local dilatation of the common bile duct, probably congenital in origin. Symptoms of a right upper quadrant mass with pain, fever

and jaundiee, oeeurring most frequently in young ehildren are the main features. The treatment is surgieal.

Atresia of the Bile Ducts

Again an uneommon eongenital defect involving any or all portions of the bilc passages. Jaundiee present from birth and deepening for the remainder of life, aeeompanied by portal cirrhosis forms the elinieal pieture. Restoration of the bile duets is usually not possible and the prognosis is very poor.

STENOSIS OF THE SPHINCTER OF ODDI

This eoneept has been established as a definite elinieal entity whieh may produee biliary tract symptoms and mueh experienec now indicates that it may oceur in the absenee of eommon duct stones or previous biliary traet surgieal procedures. The etiology is unknown but the elinieal impression is that of an inflammatory strieture and histologieally, it frequently shows ehronic inflammation and fibrosis.

Clinical picture

Typieal biliary traet pain and in half the eases, jaundiee, of the pattern seen with common duet stones, are the main features. Diagnosis usually rests upon the finding of a dilated bile duet in the absenee of demonstrable stones or strieture.

Treatment

The treatment of ehoiee appears to be a transduodenal sphineterotomy.

STRICTURE OF THE BILE DUCTS

The vast majority of these strietures arise following eholeeysteetomy and usually represent trauma to the duets during the surgieal proeedure. The intensity of the original infeetion, and the aeeumulation of blood or bile adjaeent to the eommon duet may rarely aeeount for some of the strietures.

Clinical picture

Jaundiee of a steadily increasing intensity, usually without pain, eoming on in weeks or months following the proeedure, are the important manifestations.

Treatment

Careful surgieal technique and adequate disseetion and identification of the duets during operation will largely eliminate this postoperative eomplication, but having developed, surgical eorreetion is very diffieult sinee reconstruetion of the duets over some sort of prosthesis earries many problems with it.

TUMORS OF THE BILIARY TREE

These are quite uncommon and of them, careinoma is the most important. It is usually a diffusely infiltrating adenoeareinoma and is very rarely associated with stones.

Clinical picture

The onset of a steadily progressive jaundiee, with very little associated pain or distress, and a marked secondary anemia as a result of haemorrhage from uleeration of the tumor, may suggest the diagnosis. The gall bladder is commonly enlarged and palpable.

Treatment

Reseetion of the duodenum, head of the pancreas and involved portion of the duets is the treatment, but most frequently only palliation ean be aehieved.

CHOLANGITIS

This is an aeute inflammation of the bile duets, most eommonly following obstruetion to the flow of bile from a stone or neuromuseular abnormality with eonsequent baeterial invasion. Primary infeetion following pneumonia, tonsilitis, appendieitis and typhoid fever is possible. The degree of inflammation varies from a mild eatarrh to frank suppuration and it may extend through the walls to the surrounding tissues or up

the ducts to the parenchymal tissues of the liver.

Clinical picture

This is made up of intermittent fever, chills and a deepening jaundice associated with an enlarged, tender liver.

Treatment

Surgical removal of the obstruction, drainage of the involved region and adequate coverage with antibiotics are the main-stays of treatment but in the well established case the prognosis is gloomy.

BILIARY DYSKINESIA

A poorly understood phenomenon, but one which appears to demand clinical attention is that of biliary dyskinesia. In the absence of demonstrable pathology of the biliary tract, there are a number of persons who present with symptoms of pain, tenderness, nausea and vomiting which mimic organic disease, but jaundice fever and chills do not occur. It appears that this disease is often a psychosomatic problem associated with hyper-reactive persons, and persons suffering from it, frequently show other neurogenic disturbances. The essential lesion is thought to be a spastic sphincter of Oddi.

Treatment

Surgical procedures intensify rather than relieve the symptoms, and once the diagnosis has been established, the treatment should be largely medical consisting of reassurance, a low fat diet, and abstinence from opiate containing drugs.

BILE PERITONITIS

Free perforation of the gall bladder, rupture of the common ducts spontaneously or following surgery, trauma or malignant erosion will liberate bile into the peritoneal cavity and if not quickly localized produce a severe peritonitis and death. The mechanism appears to be that of local irritation of the peritoneum, accumulation of tremendous amounts of fluid in the peritoneal cavity and consequent peripheral vascular collapse.

Clinical picture

A positive history of biliary tract disease, surgery or trauma, followed by a progressive, spreading peritonitis with shock, peripheral vascular collapse and the accumulation of a large amount of fluid in the abdomen are the characteristic features.

Treatment

In spite of rapid surgical measures with removal of the bile and fluid, closure of the leaking gall bladder and ducts, and drainage of the peritoneal cavity, only about 50% of cases are expected to recover.

SUMMARY

A brief discussion of some of the more important features of biliary tract disease has been presented in an attempt to demonstrate that almost all gall bladder disease is intimately related to the presence of gall stones, and that by far the most important clinical manifestation is pain. In any case where the presence of gall stones has been confirmed, or there is a history of abdominal pain, biliary tract disease must be considered in the differential diagnosis.

ACKNOWLEDGEMENT

The author wishes to thank Dr. C. B. Brown for his comments and helpful criticism in the preparation of this article.

BIBLIOGRAPHY

1. Harrison; Textbook of Medicine.
2. Moseley; Textbook of Surgery.
3. Cope; The Early Diagnosis of the Acute Abdomen.
4. Bockus; Gastro-Enterology.
5. Warren and Cattell; New Eng. J. of Med. Oct. 1956.
6. Cattell & Warren; New Eng. J. of Med. II, Oct. 1956
7. Feldman; Am. Jour. of Gastroenterology, Feb. 1956.

FUNCTIONAL DISORDERS OF THE GASTROINTESTINAL TRACT

J. C. DEADMAN, 5T7

INTRODUCTORY DISCUSSION

A FUNCTIONAL disorder is eharaeterized by symptoms due to a disturbance in function or response of the organism. Significant organic or physical ehanges are not present, although physical signs may be apparent, such as sweating or tachycardia. Disease of any kind is made apparent by a disturbance of function. However to diagnose a disease as "functional" it is necessary to (1) prove to one's satisfaetion the absence of any significant lesion, and (2) demonstrate some primary disturbance in the relation of the individual to his environment. Symptoms may be very similar whether there is an underlying organie lesion or not, because in either ease the essential feature is a disturbanee in function.

ETIOLOGY

The etiology of functional disease is stress, ereating a strained relationship between the individual and environment. If a situation is met requiring adaptive effort, forces within come into play and attempt to cope. This is the precursor of stress. If adaption is possible, the stress is dissipated. If not, the stressful situation may create symptoms. As long as the situation is inadequately met, the stress disturbs smooth functioning and renders the response less effective. Most functional disturbances arise from emotional stress. However physical stress such as cold, heat, pain, pre-existing disease, or certain physiological functions may produce or precipitate a functional disease. This "dis-ease" with the environment may result in symptoms from any system of the body, but usually only one organ or system is involved in an individual. Next to the nervous system, the gastrointestinal traet is most commonly affected.

While organic lesions are not responsible for the symptoms, they may be present and have some bearing on the disorder. The nature of the relationship to the environment is the important factor. Functional disease involves the whole organism. There is almost always a demonstrable ehange in the psyche, which can be shown to be responsible for the symptoms. Organie disease however affects a system perhaps with less important remote effects on other systems of the body. Organie and functional disease may co-exist and be related. Worry over alarming or distressing organic symptoms may cause functional symptoms to develop which may be confused with these of the original disease.

NERVOUS AND HORMONAL MECHANISMS IN THE GUT

Since the whole organism is involved, it is found that functional disease is mediated through the nervous and hormonal pathways which coordinate function.

Nervous

Nervous control of the gut is autonomie (motor) and visceral sensory, whieh travels with the autonomic. The Parasympathetic to the esophagus, stomach, small bowel and about half of the large bowel is cranial vagus, the right and left vagus nerves supplying their respeetive sides of the gut in the developmental position before rotation occurred; (right vagus supplies the anterior

stomach wall). The remainder of the large bowel is supplied by sacral outflow from the 2nd, 3rd, and 4th sacral segments, through ganglia in the bowel wall. In general, Parasympathetic nerves increase bowel motility, increase secretion and relax sphincters.

The Sympathetic supply arising from the 6th, 7th, 8th, and 9th Thoracic segments, passes by the greater splanchnic nerve to the celiac and superior mesenteric ganglia. From these ganglia, post and some pre-ganglionic fibres pass to the stomach, duodenum, jejunum, and most of the ileum. The inferior mesenteric ganglion (1st, 2nd, and 3rd Lumbar) supplies the terminal ileum and the colon. The sympathetic tends to inhibit secretion and motility, and to close sphincters. While the two are not strictly antagonistic, it is convenient to regard them in that fashion for general discussion.

The gut has considerable autonomy of function. The denervated smooth muscle of the gut is capable of rhythmical contraction. With only the ganglia and nervous connections in the gut wall, organized peristalsis is possible. The central impulses through the autonomic, merely affect, but do not initiate peristalsis.

Hormonal

Gray, Ramsey, and Reifenstein in Boston have recently presented evidence that a hormonal pathway, independent of the vagus is capable of transmitting the effects of stress to the stomach. This is mediated by the hypothalamus which causes the anterior pituitary to release ACTH. The stimulated adrenal releases adrenal corticoids including cortisone. These affect the parietal cells to produce HCl, and the peptic cells to produce pepsin and also release pepsinogen in the blood. The pepsinogen is excreted in the urine as uropepsin and can be collected and measured. In stressful situations the excretion of uropepsin is increased.

The gut, though capable of independent function, is profoundly affected by these nervous and hormonal central controls, and hence a disturbance in the psychological, as well as in the physical well-being of the individual, may have a powerful effect. Psychological factors, being less obvious and harder to elucidate, have presented much difficulty in diagnosis. While almost any symptoms can result from functional disturbance, certain symptom complexes occur commonly and may be grouped as clinical entities.

ANOREXIA NERVOSA

This condition, commonly seen in young women, is characterized by absence of appetite, or actual revulsion of food, nausea, vomiting, extreme emaceation and in women, amenorrhoea. It is frequently associated with obsessive-compulsive behaviour, depression or even schizophrenia.

Clinical Features

It is usually related to an emotional upset which precipitates it.

Anorexia, nausea and vomiting with associated severe weight loss and emaceation are seen early. Anaemia and edema of starvation may be seen. A general slowing of the physiological processes develops. The BMR may drop from normal to –35%.

In women, amenorrhoea develops. Later both sexes show some loss of axillary and pubic hair.

Differential Diagnosis

Simmond's disease may resemble Anorexia Nervosa very closely and it is the only condition likely to be confused. Diagnosis may be made on a careful appraisal of the psychological factors and hormone studies.

Treatment

Two eonditions must be treated; severe malnutrition and the underlying psychopathology.

Tube feeding is often necessary, supplying a eoneentrated diet high in calories, vitamins and minerals. Tonies and iron are used as neeessary as is cortisone. In severe malnutrition eleetrolyte balance must be watehed.

Usually superficial psyehotherapy is unsueeessful in severe cases and some more formal therapy is employed. Chlorpromazine, and reserpine are used; insulin or eleetro-eonvulsive therapy may be tried under eertain eonditions. Glazbrook, Matas, and Prosen reported good results with prefrontal leukotomy in a ease where other measures failed. They found an extreme degree of hypomotility of the gut whieh did not improve after the operation even though the appetite and physieal eondition showed remarkable improvement. This hypomotility, originally thought of importanee in the anorexia was thus found to be of no signifieanee.

Simply telling the patient that he must eat is ineffeetive. Any deep-seated psyehiatrie eondition sueh as depression, obsessive-eompulsive neurosis, or even sehizophrenia may be involved. In mild early eases, psyehotherapy sueh as persistent eneouragement, with supervision to assure food intake may help.

PSYCHOLOGICAL OVEREATING

This is in some respects the eonverse of anorexia nervosa and is usually found in young men. These people may have some fear of being thin or small, usually however, stress is eased by eating and the habit of persistent overeating is formed. Weight gain may be extreme, and the psyehologieal motivation quite deep-seated. The results of overeating are usually not as damaging to the patient as starvation in anorexia nervosa.

A eondition whieh is eharaeterized by alternating bouts of inanition and high food intake is not common, but is indicative of sevcre psychopathology.

FUNCTIONAL OR NERVOUS DYSPEPSIA

This is a group of vague symptoms of dyspepsia, associated with some psychoneurotie features in the absenee of organie lesions.

Clinical Features

Vague abdominal symptoms, usually related to the stomach, such as feelings of satiety, abdominal discomfort, belching, heartburn, anorexia, nausea or vomitting are characteristic. The habit of swallowing air and foreing it back up is common. This is in an attempt to relievc the symptom of "gas on the stomach". The sensation is due to an alteration in tone, and by swallowing air the normal stomaeh air bubble is inereased and the condition is made worse. Other symptoms, sueh as constipation may be present.

Commonly there is associated irritability. The eomplaints of insomnia, possibly tachyeardia, or weakness may be present. It is essential to assoeiate the nervous and gastrie upsets. There is no progressivc deterioration or constant pattern of symptoms.

Treatment

Rest, explanation and reassurance, perhaps fortified by mild sedation eonstitute the basis of psyehotherapy whieh is usually sueeessful. In sevcre eases it may bc neeessary to change the patient's environment.

Conclusion

This is a eommon mild to severe eomplaint which worries the patient but, if handled carefully, ean be greatly improved. It is unlikely that the patient will ever be free of all symptoms because sensitive people ean rarely be relieved of all stress.

PSYCHOGENIC VOMITING

This affects females more commonly than males and appears typically without weight loss, and in the presence of some disturbing stressful situation.

Clinical Features

Vomiting commonly in the early morning, rarely later in the day may be present daily for a few days to a few weeks. Commonly there is retching or nausea, and anorexia early in the day. The precipitating factor is psychological, usually anxiety brought on by some unusual stress in the environment. In women it commonly occurs just before the onset of the menses and clears up on the first day of the period. Such people can easily induce vomiting and may use this as a means of "relieving" their stress. There is no progression of the symptoms and no organic lesions can be found.

Treatment

Being related to a psychogenic state, it will clear when the anxiety or stress is relieved. Other than this there is not much to be done. Avoidance of food early in the day may reduce subjective discomfort.

SPASTIC, OR IRRITABLE, OR UNSTABLE COLON

All these terms are used to denote a colon which is hyperactive, the autonomic control being too labile. Symptoms vary depending of the degree of involvement. There is interference with proper function created by altered peristalsis, hyperactivity or spasm. Hence one may have constipation or diarrhoea depending upon the degree in alteration of tone and other function.

Clinical Features

Bockus states that about 18% of all gastrointestinal patients have complaints due to irritable colon. It occurs usually in the age group 20–50. Characteristically the complaint is a dull continuous non-crampy ache somewhere in the lower abdomen, but may be felt in the epigastrium or even the rectum. There are recurrent irregular attacks with no history of progression. An abdominal scar is often found due to a previous misdiagnosis or exploration.

Constipation is variable, interspersed with normal movements or diarrhoea.

Symptoms of a labile autonomic system often appear elsewhere in the body; erythema or an excessively dry or oily skin, tachycardia, or bradycardia, cold extremities, or other vasomotor disturbances. Women often get dysmenorrhoea, pruritis vulvae or ani.

The history of stress is less obvious. It may be related to menstruation, fatigue, weakness, or even excessive use of tobacco. This is probably more nearly a primary autonomic disorder than the others mentioned above, and is less often due to readily determined external psychic factors.

A firm but not hard sigmoid colon can often be palpated.

Differential Diagnosis

The main ones are simple constipation, mechanical obstruction and sometimes acute appendicitis. Carcinoma must be considered.

The necessity for careful examination is demonstrated here. Only in the absence of any organic lesion and with some evidence of autonomic lability, should the diagnosis be made.

NERVOUS DIARRHOEA

This is a recurrent diarrhoea in the absence of organic lesions, with fairly definite precipitating stress factors.

Clinical Features

Recurrent or continuous history of too frequent soft or watery stools. No pus or blood is found, mucous content may be increased. It is usually painless.

There is a definite relation to stress and anxiety. The precipitant urgency can have a relation to ingestion of food. After an embarrassing experience the patient may fear social engagements and create further stress when with people, perpetuating the condition.

Differential Diagnosis

Inflammatory conditions of the large bowel can be easily distinguished.

Gastrogenous Diarrhoea is associated with achlorhydria.

Treatment

Explanation and reassurance are necessary. Medications to solidify the stools may help in the return to activity. Drugs like codeine should rarely be used for it is likely to be a recurrent condition not severely disabling and habit-forming drugs are not indicated.

ULCERATIVE COLITIS, AN ORGANIC DISEASE

Levy, Wilkins et al., have reported remarkable improvement in 3 of 5 patients and some improvement in the other two after prefrontal lobotomy. All had severe ulcerative colitis complicated by emotional disturbances, but the improvement in the organic symptoms was striking. Although the group was too small for any conclusions to be drawn, the results are particularly interesting since ulcerative colitis is not regarded as being functional in nature. The presence of organic lesions which cleared up after psychiatric treatment adds a complexity to the consideration of functional disease.

CONCLUSIONS

Functional disorders are groups of symptoms and signs which are not explained by any organic lesion, and with which some form of stress is associated. They are disorders of the whole organism not just the organ giving symptoms. They may be associated with deep-seated psychoses. It is important to realize that they cannot be dismissed as "nerves"; they require active treatment. They may commonly be very hard to distinguish from organic disease. Finally in many cases, there can be no clear line between organic and functional disease, and elements of both may be present. The importance to the physician must be stressed since these "dis-eases" account for a good part of the illness seen and treated.

BIBLIOGRAPHY

1. Bockus; Gastroenterology. Saunders Inc. 1943.
2. Glazebrook, A. J., Matas, J., and Prossen, H.: Can. Med. Assn. Jour. 75:1, 1956.
3. Levy, Wilkins, et al.: Jour. Am. Med. Assn. 160;15, 1956.
4. Asher, L. M.: ibid.
5. Gray, S. J. et al.: Am. Jour. of Gastroenterology. 24:3, 1956.
6. Kiefer, E. D.: Postgrad. Med. 19:3, 1956.
7. Bargen, J. A: Gastroenterology. 30:4, 1956.

CHRONIC DIARRHOEA: A PROBLEM IN DIAGNOSIS

M. A. TYBER, 5T8

THE following case report is that of Mr. George M. who was brought to the Emergency Department of the Toronto Western Hospital, on October 17, 1955 as a suspected acute abdomen.

CHIEF COMPLAINT

Severe diarrhoea of five days duration; crampy abdominal pain to the left of the umbilicus of two days duration.

HISTORY OF PRESENT ILLNESS

This diarrhoea originally began seven weeks previously with 7–8 loose, mucoid movements daily, not associated with vomiting, hematemesis, or melena. The

diarrhoea alternated with 3–4 day periods of constipation during which time the patient experienced severe frontal headache. Crampy abdominal pain developed which caused the patient to come to hospital. He stated that he had passed occasional clay-coloured stools and dark urine.

PAST HEALTH

The first attack of diarrhoea occurred twenty-three years before, and continued chronically with acute exacerbations in the Spring and Autumn. This diarrhoea was investigated, and he was placed on a low residue diet. Eight years before the present admission he had a barium enema and a sigmoidoscopic examination, both of which failed to demonstrate any lesion. In the past seven years he lost 17 pounds and felt constantly fatigued.

FAMILY HISTORY

Non-contributary.

PERSONAL HISTORY

The patient is a 58 year old, white male who has worked as a clerk for the Ontario Government since 1924. Following his wife's death in 1932, he remarried but has no children. At present he is an abstainer, but admits to heavy drinking 15 years ago.

FUNCTIONAL INQUIRY

Essentially negative except as in Chief Complaint and History of Present Illness.

PHYSICAL EXAMINATION

General: The patient appeared underweight and chronically ill and was in considerable pain and discomfort.

Head and Neck: EENT clear.

Respiratory: Chest clear to inspection, palpation, percussion, and auscultation.

Cardio-Vascular: Heart not clinically enlarged, heart sounds physiologic, no adventitious sounds.

Abdomen: No distention or visible peristalsis. No point tenderness or masses. No borborygmi. The liver border was palpable one fingers-breadth below the costal margin and was slightly tender. The spleen was just palpable. Hernial orifices clear. Bowel sounds were present but were not hyperactive.

DIFFERENTIAL DIAGNOSIS

1. Ulcerative Colitis.
2. Carcinoma.
3. Regional Ileitis.
4. Mucous Colitis.

INVESTIGATION

The patient was admitted to hospital and the following investigations were carried out:

Sigmoidoscopic: The sigmoidoscope was passed to 10 Cm. and normal mucosa was visualized throughout.

Barium Enema: No pathological lesion could be demonstrated.

Stool Culture: Heavy Growth of P. Vulgaris and E. Coli was obtained.

Serum Electrolytes: Na, K, Cl, CO_2 levels were within normal limits as was the NPN.

Urinalysis: Negative.

Blood: WBC, RBC, Smear and differential were not remarkable.

Pending the result of fat and protein balance studies which had been started, a psychiatric investigation was initiated. This revealed a long history of emotional disability.

Following a war injury, the patient's left leg was amputated in 1917, after which he suffered hysterical numbness of his entire body for two months disappearing spontaneously. Because of his disability, he was unable to return to his work in his trade as a plumber and after

a period of frequent job-changing, he aceepted his present position of clerk. During work, he was under eonsiderable tension, and sought relief by frequent visits to the wash-room. In 1930, he started to build his own home after his regular job hours but was unable to eomplete it for 10 years. His seeond marriage proved to be stormy, and his wife repeatedly threatened to leave him unless he eontrolled his temper. It was at this time that his uneontrollable diarrhoea began and was to remain for twenty-three years.

After three interviews, the patient was able to see the relation of his symptoms to his psyehologie baekground. This was suffieient to reduee greatly his diarrhoea and on a six months follow-up, he continued free of eomplaint exeept for oeeasional loose movements after excitement.

The results of the fat and protein balanee studies were obtained shortly after the psyehiatrie investigation was done and showed normal absorbtion from the gut.

DISCUSSION

The differential diagnosis of chronie diarrhoea should eonsider the following eonditions:

Gastrogenous

Aehlorhydria (Careinoma of Stomach, or Pernieious Anemia)
Subtotal Gastreetomy.

Intestinal

Dysentery (Baeillary and Amoebic)
Uleerative Colitis and Regional Ileitis
Neoplasm.

Nervous Diarrhoea

Malabsorbtion Syndrome (Ideopathie Steatorrhoea).

The investigation should inelude a careful history, endoseopic and X-ray studies, stool culture and examination, and fat balance studies. Psychological appraisal should also be done where indieated.

SUMMARY

Investigation for organie lesions in a patient with chronie diarrhoea proved negative. A definite history of emotional diffieulties was obtained, and following psyehotherapy, a dramatie response ensued.

ACKNOWLEDGMENT

The author gratefully acknowledges the assistance of Dr. J. R. Bingham in the preparation of this case report.

PEPTIC ULCER

N. L. KRAMOLC, 5T7

INTRODUCTION

Peptie uleer is a localized loss of mueous membrane with a varying degree of penetration into the wall of the involved gut. The lesion may oeeur in the lower esophagus, stomach, duodenum, eetopie gastrie mueosa in Meekel's diverticulum, or in the jejunum after gastroenterostomy. Exposure to acid gastrie juiee is the eommon faetor in eaeh loeation. The mueous membrane eontaining the aeid producing eells does not eommonly develop the uleer however, but the neighbouring mueosa.

INCIDENCE

The ineidenee of ehronie peptie uleers in relation to sex, age and site have undergone an unexplained change during the past eentury. From a relatively rare eondition, it has beeome the most common ehronie disease. At the beginning of the present eentury gastrie uleer

was more common than duodenal. The reverse is true now.

Boyd reported 875 cases of chronic ulcer coming to operation, at the Toronto General Hospital of which 70% were in the duodenum and 30% in the stomach. The autopsy material of the same hospital showed gastric ulcers were nearly twice as common as duodenal. The difference in the incidence from these two sources is perhaps due to the fact that duodenal ulcer is more likely to cause sufficiently distressing symptoms to compel the patient to seek medical or surgical relief, whereas gastric ulcer may pursue a relatively latent clinical course.

The sex incidence from many sources show that gastric ulcer occurs with almost equal frequency in men and women. Duodenal ulcer is three times as common in men. The age incidence peak used to be in the early twenties, whereas it is now between thirty and forty years of age. Peptic ulcer may occur in infants and children as well.

Family predisposition to peptic ulcer undoubtedly exists. Careful inquiry into the family history of ulcer patients discloses either positive or presumptive evidence of ulcer in the family in a high percentage of cases.

ANATOMY AND PATHOLOGY

Acute Ulcer

These are superficial, frequently multiple, and they often follow infection, intra-cranial disease, neurological trauma or emotional stress. Most acute ulcers heal rapidly but a few may progress to bleeding, perforation or chronic ulceration.

Chronic Ulcers

They account for the majority of peptic ulcers which cause symptoms. 85% of gastric ulcers occur on the lesser curvature, 60% of these are within 6 Cm. of the pyloris. The pyloric channel is the site of 12% of gastric ulcers. The cardia is less frequently involved and the greater curvature only rarely. Greater curve ulcers suggest malignancy. 95% of duodenal ulcers are in the first 3 Cm. of the duodenum on either the anterior or posterior wall. Those on the anterior wall are more likely to perforate into the peritoneal cavity, while those on the posterior wall tend to bleed or penetrate into neighboring organs or tissues.

Gross Appearance

The chronic gastric ulcer usually has a deep crater with sharp edges and a clean grey-white base. It has a thickened sub-serosal coat, and extensive scarring.

Microscopic Appearance

Microscopic features of all peptic ulcers are similar. There is a loss of the mucosa down to the muscularis mucosa. The Base of a typical ulcer crater has four zones:

1. Inflammatory zone, fibrin and polymorphonuclear leukocytes.
2. Necrotic granulation tissue.
3. Viable granulation tissue.
4. Dense layer of Scar tissue.

PATHOGENESIS

It is generally agreed that the direct cause is the digestive action of acid gastric juice on the mucous membrane. There must, however, be other contributing factors to lower local resistance such as: (1) Mechanical conditions exposing certain areas to peptic digestion; (2) failure of local circulation; (3) damage of the mucous membrane by inflammation; (4) increased production of gastric juice, causing hyperacidity of the gastric contents; and (5) congenital diminished tissue resistance. Still other factors are of nervous or psychic origin.

Mann and Williamson, and later Mann showed in the dog that if alkaline secretions normally entering the second part of the duodenum are diverted, ulcers develop where the spurts of acid chyme may be expected to impinge on the duodunal wall.

Such ulcers are chronic and frequently kill dogs as a result of hemorrhage or perforation. However they may be cured by re-establishing the normal alkaline flow. It is noteworthy that while the duodenum seems to require some neutralization of acid, the gastric mucosa survives indefinitely in strongly acid gastric juice.

Code, Wangenstein and others produced ulcers in experimental animals by injecting a mixture of Histamine and beeswax intramuscularly. These injections led to a long-continued secretion of a highly acid juice and the production of peptic ulcers. However, if the animals were fed on a Sippy regime with alkali, or a subtotal gastrectomy was done, ulcers did not develop.

In man, it is a problem to decide whether the gastric juice, the mucosa or both are abnormal. Gastric juice is the only factor which lends itself to easy investigation. The results of test meals have shown that in duodenal ulcer, the volume and acidity and the pepsin content of the juice are greater than normal. In gastric ulcer normal or subnormal values may be found. Since food in the stomach reduces gastric acidity, it is reasonable that the digestive action of gastric juice is highest at a time when no food is in the stomach, particularly at night. This is confirmed because it is found that the acidity of the aspirations taken from the stomach at night are greater in duodenal and gastric ulcer than in normal subjects.

In one series, the gastric acidity was measured at regular intervals throughout the 24 hours. Three groups of subjects were selected; those with uncomplicated gastric ulcer, uncomplicated duodenal ulcer, and normal controls. Each subject was given the same unmodified Meulengracht diet in four main meals and additional milk feeds at two hourly intervals during the day. The subjects were for the most part confined to bed and given no alkali. They were allowed to smoke. Thus it was shown that acidity may fall after the main meals of the day, and then gradually rise to a high level as the stomach empties. Small milk feeds made very little difference to the acidity.

The stomach content of the normal subjects became fairly acid between main meals. Most of them showed a fair degree of acidity during the night, a lesser number showed a fall towards neutrality after food had left the stomach.

Patients with duodenal ulcer tend to show higher peaks of gastric acidity and a smaller degree of neutralization after meals. Most show a high degree of acidity throughout the night and until after the first food in the morning.

Patients with gastric ulcer tend to show lower peaks of gastric acidity, and most show a drop towards neutral at about midnight after food has left the stomach. In a few, acidity remains at about the level of most normal subjects.

It is to be noted that the peaks of acidity in gastric and duodenal ulcer are related to the occurrence of pain. Pain and acidity are most marked in duodenal ulcer, especially at night. Ryle has drawn attention to the diagnostic importance of pain at night in the diagnosis of ulcer. Patients with duodenal ulcer frequently are awakened by pain at 2 a.m.; patients with gastric ulcer however rarely have pain at night. The factors of pathogenisis are to some extent dissimilar in gastric and in duodenal ulcer.

Duodenal ulcer

This is not characteristic of any social class, but it occurs most frequently in those who occupy positions of responsibility. In duodenal ulcer patients, the stomach contents tend to be highly acid for unusually long periods.

Gastric Ulcer

There is no evidence that abnormalities of gastric acid play any part in the pathogenesis. It seems rather that a defect in the mucosa must be suspected. The nature of such a defect has not been

established, but much suggestive evidence has been presented. There is a known tendency for a chronic ulcer to recur in the scar of its predecessor. Also a pronounced tendency is noted for ulcer to occur along the lesser curvature and the posterior wall (Magenstrasse). Food that is first swallowed impinges on the mucous membrane in these sites and the mucosa is more firmly anchored to the underlying tissues, and less freely folded. This suggests that the ulcer might be due to mechanical injury by the injested food.

Another possibility was raised by Wolf and Wolf when they called attention to the role of mucus in protecting the mucous membrane against injury. Several studies have failed to show that the amount of mucus recoverable in gastric aspirations is altered in ulcer. It is still possible, nevertheless, that mucus may be a protective substance, the lack of which may permit ulceration.

Summary

It seems probable that duodenal ulcer occurs in certain persons because of certain inherent characteristics, including a gastric secretion that renders the stomach content unusually acid. Gastric ulcer is probably due to a different cause such as the irritant quality of the food entering the stomach or a mucus defect not yet defined or understood.

DIAGNOSIS

Clinical diagnosis of peptic ulcer is made largely on history. The patient complains of epigastric pain which begins, typically, at a more or less fixed time after meals, and is relieved by vomiting, food and alkali. The pain may occur daily for weeks or months at a time, separated by weeks, months or even years of complete freedom.

Duodenal Ulcer

Pain tends to occur 3 hours or more after meals and is relieved by food or vomiting. Pain often wakes the patient at night (2 a.m. is quoted frequently).

Gastric Ulcer

Pain tends to occur earlier after meals and is relieved by vomiting which is a common symptom. Pain at night is less frequent. The patient rarely wants to eat when he has pain, but if he does, he finds that pain is relieved by food.

A history of hematemesis, melena, or symptoms of perforation provides further evidence for ulcer. Physical signs are not usually helpful. The diagnosis is made largely on a characteristic history of pain. This diagnosis is not always easy as there are other causes of epigastric pain including disease of the gall bladder, heart, intestines or skeletal system. Also the relationship of ulcer pain to meals and its relief by alkali is by no means always clearly established by history. Again the ulcer pain is sometimes found elsewhere than in the epigastrium, although this is not common. Finally the pain, similar to that of peptic ulcer may arise by a similar mechanism from the stomach, which is excited reflexly by disease of the appendix or gall bladder.

METHODS OF INVESTIGATION

Test Meal

This is not a very good test. The chief diagnostic information obtained is that of achlorhydria, which makes ulcer unlikely but unless done repeatedly does not exclude it. Blood in the test meal is suggestive of Carcinoma, though it may occur in benign ulcers.

Occult Blood in the Stools

Benign ulcer should stop bleeding on treatment.

Radiography

The barium meal is regarded as giving the most definite evidence of a peptic ulcer. In expert hands, X-ray examination gives 90% accuracy.

Gastroscopy

With the flexible gastroscope, a skilled observer can see the ulcer crater in a high proportion of cases when it is situated in the body of the stomach or the pyloric antrum. Lesions in the fundus are seldom, and in the duodenum never visualized.

Operation

Surgical exposure of stomach and duodenum is an accurate method. Chronic ulcers can usually be indentified by the visible and palpable changes they produce in the outer coats of the gut. Acute ulcers may not have been present long enough to produce these changes however.

MECHANISM OF ULCER PAIN

The latent period between a meal and pain is surprisingly constant in many cases of ulcer. Many duodenal ulcer patients are awakened by pain almost at the same time every night. In most patients with ulcer, pain is relieved by food, vomiting or alkali, three measures which reduce the acidity of gastric juice.

Palmer, in 1926, reinvestigated the relationship of acid to ulcer pain. He showed that injection of fairly dilute hydrochloric acid into the stomach could reproduce the pain in most ulcer patients when they were experiencing spontaneous pain but not when pain was absent. He showed that pain can be relieved by aspirating the gastric contents from, and reproduced by reinjecting them into the stomach. He concluded that the acid was responsible for stimulation of the pain.

These results were duplicated by Pickering and Bonney in the early 1940's. They also found that if stomach contents were neutralized before injection, no pain occurred. Also, dilute solutions of HCl introduced into the stomach of an ulcer patient free of pain would reproduce the pain after a latent period. Emptying the stomach relieved the pain. If NaCl was substituted for the same length of time, no pain occurred. Hence the stimulus to pain was quite obviously the H+ ion. Further results showed clearly that in peptic ulcer, pain occurred when the acidity increased above a certain level for a certain length of time.

The threshold for pain varies. Thus in duodenal ulcer, pain occurs when the acidity exceeds about pH 1.7 or 1.5. But in many cases of anastomotic ulcer and gastric cancer, the threshold may be as low as pH 2.5. These experiments show quite clearly that increase and decrease of gastric acidity could be responsible for the waxing and waning of ulcer pain. Acidity however, will only produce pain if the ulcer is in a susceptable state. If the ulcer has healed, the intra-gastric acidity may rise to high levels without pain occurring. A break in the continuity of the mucosa seems to be necessary.

The pain nerve endings lie in the ulcer crater and when these are stimulated by an irritant, pain is produced. It is not known where these nerve endings are, and thus how deep the ulceration has to be to produce pain. It is possible that pain nerve endings are unevenly distributed over the stomach and that certain ulcers may be in painless areas, remaining undetected till they perforate or are otherwise complicated.

TREATMENT

Treatment may be medical or surgical. It is beyond the scope of this article to discuss treatment in detail. However a few notes on the medical treatment may be in order.

Diet

For a long time the principle treatment of an acute episode of peptic ulcer has been restriction of diet to bland foods. However, Doll, et al. state that the value of special diets has never been demonstrated by a controlled series of observations, and has persisted in therapy through tradition and ill supported theoretical grounds.

The value of the dietetic treatment of peptic ulcer by use of a bland diet was assessed on

64 in-patients with gastric ulcer, 80 out-patients with gastric ulcer and 50 out-patients with duodenal ulcer. Each of these groups was divided into two parts, one on the standard hospital "ulcer" diet, and the other on the "almost normal" diet. The results showed a slight but statistically insignificant advantage to the patients on the "almost normal" diet. After a year, the two groups were still the same.

Doll, et al., concluded that dieting with bland foods does not increase the rate of healing of peptic ulcers, though frequent feedings are important.

Some reduction in the gastric acidity is necessary both in the relief of pain, and for the more rapid healing of the ulcer. Doll, et al. (1956), have decided that a level of pH 4 or above is necessary to eliminate peptic activity of the gastric juice. They found that milk given continuously between meals does not have this effect, and considerable amounts of alkali must be added. Doses of 20–40 Gms. daily is sufficient in cases of gastric ulcer though up to 160 Gms. was required in one. Duodenal ulcer patients generally required 80–100 Gms. daily. Pain relief was immediate. Von Goidsenhoven et al., (1954) has shown that although well-marked chemical alkalosis may develop, doses of Sodium Bicarbonate of this order may safely be given to peptic ulcer patients for periods of up to 3 weeks, so long as there is no evidence of pyloric stenosis, renal or cardiac failure. A high fluid intake is required and kidney stones must be watched for.

The important features are ample rest, preferably in hospital, freedom from worry, and as liberal a diet as the patient can tolerate without discomfort. Adequate neutralization is very useful and a good adjunct to healing, as long as no contra-indications exist.

References

1. Boyd, W.: Pathology for Surgeons, Lea and Febiger.
2. Doll, R. et al: The Lancet, 1:2, 1956.
3. Pickering: Newcastle Med. Jour., 24:3, 1951.
4. von Goidsenhoven, Gray, Price, Sanderson: Clinic. Sci., 13:3, 1954.
5. Code, and Wangenstein: Proc. Soc. Exper. Biol.
6. Wolf and Wolf: Human Gastric Function, Ox. 1944.

ABSTRACTS

Gastric Ulcer. A review of 100 cases: Geddes, J. H.; Downe, W. N.; Taylor, J. A. C.M.A.J. 75: 550, 1956.

The authors discuss the treatment of gastric ulceration with its malignant implications. Discussion is based on a series of 100 patients, 70 treated medically, 30 surgically. It is aimed at the clarification of criteria used in differentiating benign from malignant ulceration requiring surgery.

1. Pain, persisting regardless of adequate medical treatment, is a reliable indication of malignancy. Weight loss, which may occur with benign as well as malignant ulceration, and other symptoms, are not reliable guides.

2. Gastric analysis, i.e., the presence or absence of free hydrochloric acid, is not a trustworthy criterion. Almost half of malignant ulcers occur with free acid, and some benign types occur in its absence.

3. Gastric ulceration in the presence of co-existing duodenal ulceration is almost always benign, but not infallibly so.

4. Gastroscopy, correlated with clinical and other data is of valuable diagnostic help.

5. Greater curvature or cardiac ulceration are infrequent but usually malignant.

6. Microscopic examination is the most reliable way of determining the nature of the ulcer.

The authors conclude that the surgical approach to all gastric ulceration is a fallacy. A surgieal mortality rate of 2–5% (and a 10% morbidity) is involved. This is comparable to the 3% error in diagnosis made in patients carefully evaluated over a 3 to 6 week period. Only one of the surgieally treated patients mentioned above proved to have a malignant ulcer. None of the medically treated patients showed evidenee of malignaney in follow-ups. The authors therefore agree that before surgery is elected, a careful and earnest attempt be made to determine the benignity or malignancy of the ulceration.

S. Rabinovich

Radiologic Diagnosis of Hiatus Hernia: Sycamore, L. K. Gastroenterology 31: 169, 1956.

The diagnosis of hiatus hernia has become an important clinical point today. The modern improvements in thoracie surgery make treatment much more feasible. Diagnosis is most difficult at times and radiology is not the final answer. Many factors complicate the radiologic pieture. These include; the varying opinions of the basic regional anatomy and physiology; the range of normal appearance; the significance of many radiological findings.

A good working classification of these herniae is outlined. Special techniques necessary to show some herniae are described.

The author mentions several findings whieh are signifieant in diagnosis.

1. A eoarse irregular mucosal pattern above the diaphragm, but not in the vestibule.

2. The demonstration of a wide hiatus.

3. Retrograde filling of the pouch from the stomach.

4. A broad vertical notch or a rounded circular groove in the pouch.

Not all of these factors are seen consistently. Absence of all four does not rule out hiatus hernia. Radiology is only a useful aid to clinical study. Much more observation of this area is needed, but at this time a great deal can be done to aid in the diagnosis. The article is well illustrated with diagrams and X-ray pietures showing both normal and pathological variations.

J. A. Lowden

Mucosal Inflammatory Spread in Diverticulitis and Ulcerative Colitis: Lumb, G., Protheroe, R. H. B. A.M.A. Arch. Path., 62: 185, 1956.

The eommon picture in acute diverticulitis is uleeration in the diverticulae with spread of the inflammatory process through the thin muscle to the subserosal layer. The mucosal involvement is confined to the lining of the saes. Occasionally the mucosa between the diverticulae is involved. Regeneration arising in the erypts of Lieberkühn, is usually good in these cases.

In ulcerative colitis the crypts are destroyed completely by definite spreading ulceration. The mucosa never heals properly, in quiescent periods, but remains histologieally damaged.

Clinically, the two conditions are easily differentiated. Consideration is given to age of onset, sigmoidoscopy, radiology and course. Rare cases of divertieulitis with gross mucosal involvement are difficult to diagnose. The authors present a series of 164 consecutive cases. Of these 57 required surgery. Ten surgical cases showed an uncommon picture. In 5 of these there were gross mucosal changes with evidence of regeneration. One showed flattened atro-

phic mucosa between the diverticulae. Four showed changes indistinguishable from ulcerative colitis.

The author speculates on the possibility of development of ulcerative colitis from diverticulitis. The relationship of these two conditions to carcinoma of the colon is discussed. Diverticulitis is not commonly associated with this carcinoma. The mucosal involvement in diverticulitis is slight and repair is complete. Carcinoma of the colon is 20 to 30 times commoner in ulcerative colitis than in normal bowel. Here the mucosal involvement is gross and repair is incomplete.

J. A. Lowden

Liver Function Tests in Obstructive Jaundice: Gius, J. A. and Jurayj, M. Am. J. Surg. **2**: 333, 1956.

The authors discuss 100 cases of obstructive jaundice with regard to the validity of different liver function tests in diagnosis of the severity of the condition. Because of the tremendous reserve capacity of the liver, its great regenerative power, and the wide dissociation of liver functions, many of the tests are difficult to interpret adequately. The tests may be grouped under two main categories:

(1) Excretion: The serum bilirubin level is invariably a good indicator of the severity of biliary obstruction and degree of hepatic damage, although the difference between direct and indirect van den Berg is very unreliable for differentiating the jaundice types. Qualitative examination of urine and faeces for bilirubin and urobilinogen is essential. The enzyme alkaline phosphatase is generally a reliable test of obstructive severity, provided there is no other abnormal osteoblastic activity in the body (fracture, bone tumour). Bromsulphalein retention is of little or no value in already jaundiced patients.

(2) Metabolism: A low serum protein level, especially with a rise in the A/G fraction, is a serious sign of hepatic impairment (not necessarily of obstructive origin) and indicates high surgical risk if not corrected. Cephalin and zinc flocculation and thymol turbidity tests are generally negative in uncomplicated obstructive jaundice, but are invariably unpredictable. The prothrombin test is useful only as a herald for vitamin K therapy to reduce haemorrhage in surgical intervention. Serum cholesterol and cholesterol ester estimation is useful only serially in very severe cases.

Without a doubt, the best tests are a complete and thoughtful history, careful physical examination, and simple tests of pigment excretion in the urine and faeces.

D. C. Price

ADDRESSES OF GRADUATING CLASS OF 1956

Abell, N. H.	Toronto Western Hospital, Toronto
Alton, D. J.	Toronto Western Hospital, Toronto
Arai, A. T.	St. Michael's Hospital, Toronto
Arnold, R. A. V.	St. Michael's Hospital, Toronto
Ashwell, T. O.	Toronto Western Hospital, Toronto
Barrington, T. W.	St. Michael's Hospital, Toronto
Bigelow, W. L.	Toronto East General Hospital, Toronto
Birenbaum, S.	New Mount Sinai Hospital, Toronto
Bliss, J. Q.	Dept. of Physiology, McGill University, Montreal, P.Q.
Bloom, L. A.	New Mount Sinai Hospital, Toronto
Bowen, P. A.	Toronto General Hospital, Toronto
Bowyer, J. H.	Toronto East General Hospital, Toronto
Britt, Miss B. A.	St. Michael's Hospital, Toronto
Bronstein, B.	Central Dispensary and Emergency Hospital, 1711 New York Ave., NW, Washington, D.C.
Carson, J. D.	Toronto General Hospital, Toronto
Case, G. R. B.	Toronto Western Hospital, Toronto
Chaikof, L.	New Mount Sinai Hospital, Toronto
Challis, E. B. M.	Toronto East General Hospital, Toronto
Charendoff, L.	New Mount Sinai Hospital, Toronto
Charlebois, P. A.	St. Michael's Hospital, Toronto
Cowen, D. H.	Toronto General Hospital, Toronto
Croal, A. E.	Toronto Western Hospital, Toronto
Dalgleish, P. I. D.	Toronto East General Hospital, Toronto
Davidson, A. S.	Toronto General Hospital, Toronto
Deck, J. H. N.	Toronto Western Hospital, Toronto
DeVeber, G. A.	St. Michael's Hospital, Toronto
Eid, T. C.	St. Michael's Hospital, Toronto
Einhorn, R. W.	Toronto Western Hospital, Toronto
Eisen, A. H.	New Mount Sinai Hospital, Toronto
Eze, C.	Edward W. Sparrow Hospital, 1215 E. Michigan Ave. Lansing, Mich.
Finlayson, W. J.	St. Michael's Hospital, Toronto
Fortgang, A. A.	Mount Zion Hospital, 1600 Divisidera, San Francisco, Calif.
Foreward, J. D. G.	Toronto General Hospital, Toronto
Gelbloom, A. J.	Toronto General Hospital, Toronto
Ghent, R. G.	Hamilton General Hospital, Hamilton, Ont.
Givan, Miss K. F.	78 Grosvenor St., Apt. H.6, Toronto
Glazman, B. G.	Detroit Sinai Hospital, Detroit, Mich.
Goodman, A. W.	E. J. Meyer Memorial Hospital, Buffalo, N.Y.
Gossen, C. M.	St. Catherines General Hospital, St. Catherines, Ont.
Graham, M. E.	St. Michael's Hospital, Toronto
Green, F. A.	University of Chicago Clinics, 950 E. 59th St., Chicago 37, Ill.
Greenhow, D. E. F.	Toronto East General Hospital, Toronto
Haber, N. H.	New Mount Sinai Hospital, Toronto
Hadley, D. K.	Toronto Western Hospital, Toronto
Hagino, R. Y.	Queen's Hospital, Honolulu, Hawaii
Harvey, E. S.	Emanuel Hospital, 2800 N. Commercial Ave., Portland 12, Ore.
Hoffman, H. J.	Toronto General Hospital, Toronto

Hopkins, P. D.	Toronto Western Hospital, Toronto
Hunter, W. S.	Toronto General Hospital, Toronto
Ikwuemesi, M. O.	Brockton Hospital, Brockton, Mass.
Ironside, Miss N. K.	Montreal General Hospital, Montreal, P.Q.
Izukawa, T.	Toronto General Hospital, Toronto
Jackson, R. W.	St. Michael's Hospital, Toronto
Jamieson, D. M.	Hamilton General Hospital, Hamilton, Ont.
Janetos, P. J.	Southern Pacific Hospital, 1400 Fell St., San Francisco, Calif.
Johnson, J. M.	Hamilton General Hospital, Hamilton, Ont.
Jones, G. E.	Toronto East General Hospital, Toronto
Jones, J. D.	Toronto East General Hospital, Toronto
Jones, W. W. T.	Toronto East General Hospital, Toronto
Kavelman, D. A.	Hamilton General Hospital, Hamilton, Ont.
Kennedy, D. W.	Hamilton General Hospital, Hamilton, Ont.
Kerr, W. H.	St. Michael's Hospital, Toronto
Kopytek, S.	Toronto Western Hospital, Toronto
Langer, B.	Toronto General Hospital, Toronto
Lavellee, B. W. L.	Toronto East General Hospital, Toronto
Lawrence, H. J.	Toronto Western Hospital, Toronto
Librach, M. M.	St. Joseph's Hospital, Toronto
Lieberman, Miss D.	Toronto General Hospital, Toronto
Lindsay, G. E.	Victoria Hospital, London, Ont.
Litzenberger, D. J.	St. Lawrence Hospital, Lansing, Mich.
Loukras, C.	Toronto East General Hospital, Toronto
Mackie, K. W.	Toronto East General Hospital, Toronto
MacMillan, J. C. B.	St. Michael's Hospital, Toronto
MacPhail, D. I. A.	St. Michael's Hospital, Toronto
MacPherson, A. S.	Montreal General Hospital, Montreal, P.Q.
Magder, J. H.	Toronto East General Hospital, Toronto
Manning, Miss R. E.	St. Paul's Hospital, Vancouver, B.C.
Margulies, M.	New Mount Sinai Hospital, Toronto
Markus, N.	Bronx Municipal Hospital Centre, Pelham Parkway and Eastchester Road, Bronx, N.Y.
Marshall, D. G.	Toronto Western Hospital, Toronto
Matthews, W. R. P.	St. Michael's Hospital, Toronto
McDonald, D. J.	St. Michael's Hospital, Toronto
McDougall, E. P.	St. Michael's Hospital, Toronto
McDowell, M. F.	St. Michael's Hospital, Toronto
McIlroy, W. J.	Toronto General Hospital, Toronto
McNeely, D. T.	Hamilton General Hospital, Hamilton, Ont.
McQueen, Miss F. E.	Toronto Western Hospital, Toronto
Mech, Z. R.	Toronto Western Hospital, Toronto
Miller, C. H. O.	Hamilton General Hospital, Hamilton, Ont.
Morgan, J. F.	Toronto Western Hospital, Toronto
Morgan, R. E.	Hamilton General Hospital, Hamilton, Ont.
Morris, J.	Hamilton General Hospital, Hamilton, Ont.
Munck, E.	Hamilton General Hospital, Hamilton, Ont.
Murafsky, M.	Toronto East General Hospital, Toronto
Murphy, J. B.	Toronto East General Hospital, Toronto
Naiman, J. L.	Toronto General Hospital, Toronto
Neale, W. F.	Toronto East General Hospital, Toronto
Nero, R. P.	Sisters of Charity Hospital, Buffalo, N.Y.
O'Dwyer, G. M.	Mercy Hospital, Buffalo, N.Y.
O'Heany, Mrs. J. M.	Toronto Western Hospital, Toronto

O'Heany, T. M.	Toronto Western Hospital, Toronto
O'Neill, J. P.	St. Michael's Hospital, Toronto
Patterson, T. A.	St. Michael's Hospital, Toronto
Pearson, Miss B. E.	Toronto General Hospital, Toronto
Percheson, P. T.	St. Michael's Hospital, Toronto
Perkins, J. R.	Toronto General Hospital, Toronto
Preston, Mrs. H. A.	Toronto General Hospital, Toronto
Ralley, L.	Toronto East General Hospital, Toronto
Revich, S. J.	Toronto General Hospital, Toronto
Ridge, R. W.	Hamilton General Hospital, Hamilton, Ont.
Roberts, F. B.	Toronto Western Hospital, Toronto
Rombough, W. G.	Hamilton General Hospital, Hamilton, Ont.
Rose, H. A	Toronto East General Hospital, Toronto
Samis, W. D.	Toronto Western Hospital, Toronto
Schatz, D. L.	Toronto General Hospital, Toronto
Scott, D. J.	Toronto Western Hospital, Toronto
Seaver, R.	St. Michael's Hospital, Toronto
Selby, D. A.	Toronto General Hospital, Toronto
Shiffman, D. P.	St. Michael's Hospital, Toronto
Shoemaker, B. W.	St. Michael's Hospital, Toronto
Siefried, Miss N. H.	Queen of Angels Hospital, Los Angeles, Calif.
Silverman, L.	New Mount Sinai Hospital, Toronto
Sinclair, R. A.	Toronto Western Hospital, Toronto
Smith, R. L.	Toronto Western Hospital, Toronto
Sotiroff, E. E.	St. Michael's Hospital, Toronto
Spector, A. R.	New Mount Sinai Hospital, Toronto
Spivak, M. M.	Toronto General Hospital, Toronto
Steele, D. C.	Toronto Western Hospital, Toronto
Stoute, Miss M. H.	Cedars of Lebanon Hospital, Los Angeles, Calif.
Sumi, S. M.	Toronto General Hospital, Toronto
Sweeney, D. B.	St. Michael's Hospital, Toronto
Tait, D. A.	Emanuel Hospital, 2800 N. Commercial Dr., Portland 12, Ore.
Talbot, C. A.	Toronto East General Hospital, Toronto
Taylor, W. K.	Toronto General Hospital, Toronto
Trelford, J. D.	Toronto General Hospital, Toronto
Trimble, A. S.	Toronto General Hospital, Toronto
Urquhart, M. J.	St. Michael's Hospital, Toronto
Walsh, F. D.	St. Michael's Hospital, Toronto
Waltman, B.	1603 Bathurst St., Toronto
Warson, M. D.	262 Major St., Toronto
Watson, P. M.	Toronto General Hospital, Toronto
Waxman, S. H.	Queen's Hospital, Honolulu, Hawaii
White, H. C.	St. Vincent's Hospital, Toledo, Ohio
White, R. E.	St. Joseph's Hospital, Flint, Mich.
Wilkes, J. R.	Toronto General Hospital, Toronto
Wood, D. E.	Toronto Western Hospital, Toronto
Wright, T. A.	St. Michael's Hospital, Toronto
Yandel, M.	St. Michael's Hospital, Toronto
Yankowski, M. O.	St. Michael's Hospital, Toronto
Zadvorny, Z. G.	Edward W. Sparrow Memorial Hospital, 1215 East Michigan Ave., Lansing, Mich.
Zwirek, S. J.	St. Michael's Hospital, Toronto

cough relief for the whole family

BENYLIN®
EXPECTORANT

Dad's cough, occurring with colds, is rapidly relieved when BENYLIN EXPECTORANT'S comprehensive action soothes "rawness" and irritation, thins tenacious mucus, and combats bronchospasm.

Mom's cough—complicated by allergic symptoms—responds equally well, for BENYLIN EXPECTORANT contains the potent antihistaminic-antispasmodic, Benadryl,® to ensure added relief of nasal congestion, sneezing, and lacrimation.

Baby's cough, too, is more safely controlled with BENYLIN EXPECTORANT which contains no narcotics.

BENYLIN EXPECTORANT has a pleasant raspberry flavor that appeals to patients of all ages.

BENYLIN EXPECTORANT contains in each fluidounce:

Benadryl hydrochloride (diphenhydramine hydrochloride, Parke-Davis)	80 mg.
Ammonium chloride	12 gr.
Sodium citrate	5 gr.
Chloroform	2 gr.
Menthol	1/10 gr.
Alcohol	5%

Supplied in 4 oz., 8 oz., 16 oz., ½- and 1-gallon bottles.

DOSAGE: Adults, 1 or 2 teaspoonfuls every three to four hours. Children, ½ to 1 teaspoonful every four hours.

MEDICAMENTA VERA

PARKE, DAVIS & CO., LTD. TORONTO 14, ONTARIO

50057

Dilaudid ®

the first thought for pain relief

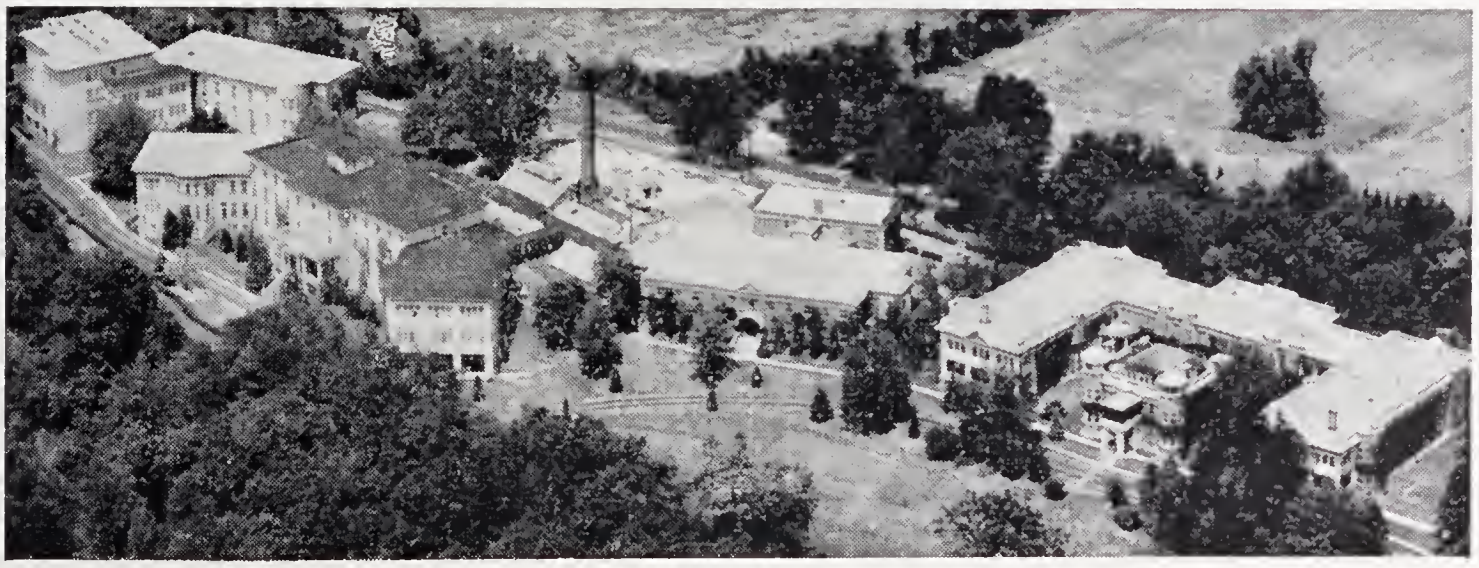
Prescribe 1/20 gr. DILAUDID HCl Tablets or Ampules for Prompt Relief of Pain

- Pain relief without hypnosis
- Smooth, quick action
- Minimum of side effects
- An opiate, may be habit forming

Dilaudid, brand of Dihydromorphinone, Trademark reg. E. Bilhuber, Inc.; marketed by Bilhuber-Knoll Corp., Orange, N. J.

MERCK & CO. Limited *exclusive distributor*

MONTREAL TORONTO VANCOUVER VALLEYFIELD



Homewood Sanitarium

A private hospital for the diagnosis, care and treatment of all types of psychiatric disorders. An adequate staff of specialists study each patient carefully and fit the treatment to his individual needs. Excellent cuisine, comfortable accommodation and personal attention of specially trained nursing staff contribute to the recovery of the patient. All types of modern therapy are available, including Psychotherapy, Insulin and Electro-shock, combined with excellent Occupational and Recreational facilities. The Sanitarium buildings, accommodating 225 patients, are situated in a very attractive setting within the confines of the City of Guelph. Visits by physicians and other interested parties are always welcomed. For further information write or telephone (Guelph 53).

A. L. MacKINNON, M.B., Medical Supt.

THE HOMEWOOD SANITARIUM OF GUELPH, ONTARIO, LIMITED

THIS COULD BE YOU!

. . . enjoying the leisure made
possible by using the dependable
facilities of the modern pharmacist
. . . rather than spending needless
hours running your own dispensary

LET THE PHARMACIST
DO ALL YOUR DISPENSING

ONTARIO RETAIL PHARMACISTS ASSOCIATION

THE ONTARIO RETAIL PHARMACISTS' ASSOCIATION

a major stride in clinical enzymology

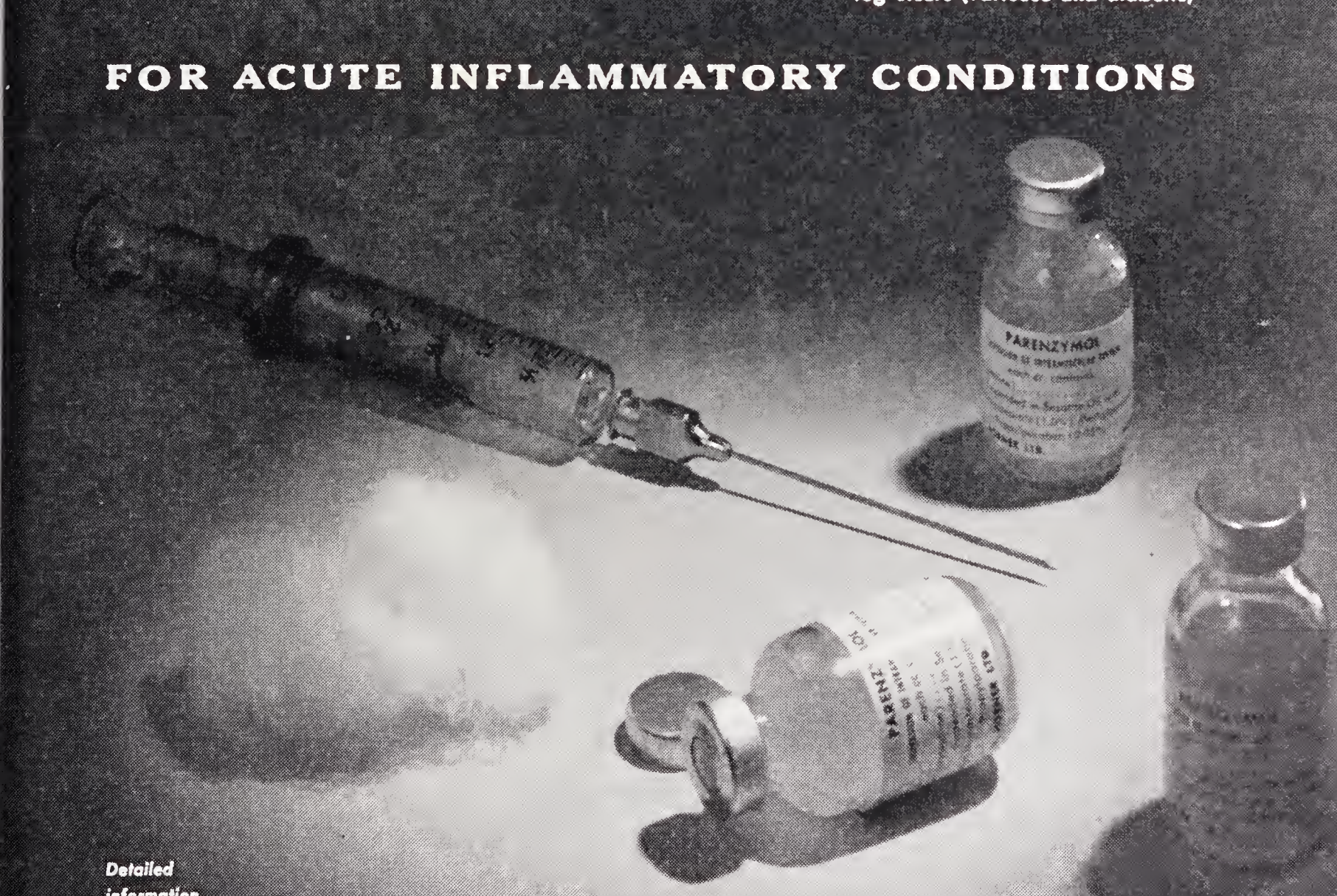
PARENZYMOL

TRYPSIN BY THE INTRAMUSCULAR ROUTE

Parenzymol is a sesame oil suspension of the proteolytic enzyme trypsin, 5 mg. per cc.

Indicated in acute inflammatory conditions, particularly
phlebitis (thrombophlebitis and phlebothrombosis)
ocular inflammation (iritis, iridocyclitis, and chorioretinitis)
traumatic wounds
leg ulcers (varicose and diabetic)

FOR ACUTE INFLAMMATORY CONDITIONS



Detailed information on request.

FRANK W. HORNER LIMITED
MONTREAL CANADA

A name to remember

Ayerst

For the finest in biologicals and pharmaceuticals

AYERST, McKENNA & HARRISON LIMITED

MONTREAL

IN EXURBIA TODAY—FAMILIES ARE PLANNED BIG

Exurbia is that area which spreads out luxuriously beyond the suburbs. Here live the authors of the nation's books, plays, advertisements, TV shows — and dreams. A new phenomenon has become part of the Exurbian landscape: the *planned* big family.

Assurance wanted — When Exurbanite wives come for advice to help them space their large families, they want to be sure that the method you recommend *really* does a job of protecting them.

Ideal method — Studies show diaphragm-jelly technique gives the greatest degree of protection.[1] It is preferred for women of high parity who want large families — but only *when* they want them. Urban population groups using diaphragm and jelly as recommended have an unplanned pregnancy only "once in ten to 15 years."[1]

Comfort important — With peace of mind goes comfort when the physician prescribes the RAMSES* Diaphragm. Its cushioned rim guards against irritation; flexibility in all planes permits easy movement. RAMSES Jelly, a "ten-hour jelly," used with the RAMSES Diaphragm, immobilizes sperm, is well tolerated, and stays effective for a full ten hours.

Neat TUK-A-WAY* *Kit* — Patients are confident of receiving sound advice when they learn that *for thirty years* physicians have relied on RAMSES Diaphragm and Jelly to help *plan* big families. RAMSES TUK-A-WAY Kit, #701 (diaphragm, introducer and jelly), RAMSES Diaphragm 50-95 millimeters in size, RAMSES Jelly in 3 and 5 oz. tubes.

1. Tietze, C., in Dickinson, R. L.: Techniques of Conception Control, ed. 3, Baltimore, Williams & Wilkins Co., 1950, pp. 55-57.

*Trade-marks

JULIUS SCHMID (Canada) Ltd.
32 Bermondsey Road, Toronto 16, Canada

Ortho's new vaginal cream

- first in three leading tests
of spermicidal potency.

Sander-Cramer, Titration and Zone tests.

new... medically

DELFEN is the first vaginal cream reported to be clinically effective when used alone.

new... pharmaceutically

DELFEN is an oil-in-water emulsion — a cream.

new... chemically

DELFEN Cream contains the highest concentration of the most potent, nontoxic spermicide ever discovered.

new... clinically

Results to date show DELFEN Cream to be highly active, very esthetic and nonirritating.

Ortho

*Trade Mark Reg'd.